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ARCHIVES OF OTOLARYNGOLOGY

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CHANGES OF THE TEMPORAL BONE IN LEUKEMIA AND OSTEITIS FIBROSA

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CHICAGO

The temporal bone differs from other parts of the skeleton in two respects: It is not subject to direct mechanical influence, and it has anatomic structures peculiar to itself.

To understand better the pathologic changes within the temporal bone one needs to review its anatomy briefly. The most interesting part of the bone from the pathologic standpoint is that which encloses the membranous internal ear. This is called the "pyramid of the temporal bone" or the "capsule of the internal ear." The present study deals chiefly with this aspect of the temporal bone.

STRUCTURE OF THE BONY CAPSULE OF THE INTERNAL EAR

The bony capsule of the internal ear in adults consists of (*a*) the enchondral layer, (*b*) the periosteal layer and (*c*) the endosteal layer.

Enchondral Layer.—This layer develops by enchondral ossification of the cartilaginous anlage of the bony capsule. In contradistinction to the enchondral ossification of the long bones, osteoclasts do not play an important part in the development of this layer. There is rather a persistent progressive formation of new bone without interference of definite resorption of bone. The continued formation of new bone leads to the development of an ivory-like compacta which is completed by the end of the second year of postfetal life. At this stage the enchondral layer consists of bone formed by strands of parallel fibers and not by lamellas—a type of bone which makes only a transitory appearance in the development of long bones but is a permanent feature of the enchondral layer.

In the mature enchondral layer cartilaginous interglobular spaces are to be found containing calcified chondroid tissue but not hyaline cartilage—a type of tissue which behaves similarly to the strandlike bone mentioned in the foregoing paragraph. There are, further, blood vessels, the walls of which contain a thick sheath of basophilic substance, and small marrow spaces, which are seen only occasionally and the boundaries of which are always aplastic. Cement lines are absent.

Following the second year of life, the enchondral layer undergoes gradual atrophy. The osteocytes become invisible, the sheaths of the blood vessels become thicker and the lumen is gradually obliterated. Thus the enchondral layer of the adult presents markedly decreased vitality and metabolism.

Periosteal Layer.—By primary ossification of connective tissue, this layer develops within the periosteum including the dura. It forms a network of woven bone. Strandlike bone is deposited in the meshes of this network. Later lamellar

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bone appears around the blood vessels forming haversian systems. In adults this layer consists of bone with either a plexus or a strandlike structure, occasionally a lamellar structure, and of marrow spaces containing either fatty substance or a lymphoid or mixed marrow, the boundaries of which either are aplastic or present inconspicuous signs of deposition and absorption of bone. As the capsule of the internal ear grows exclusively by periosteal deposition of bone after the second year of life, the periosteal layer presents vital processes consisting of absorption and deposition of bone during the entire span of life, even during senility.

Endosteal Layer.—This layer develops within the endosteum by primary ossification of connective tissue. It is by no means as well developed as the periosteal layer. It plays a minor role in the pathology of the capsule. It is best developed in the modiolus of the cochlea and in the lamina cribrosa of the maculas and the cristae. It consists of woven bone and of channels which contain blood vessels and connective tissue, particularly in the modiolus.

The three layers can be recognized easily in the area of the cochlea. At the tip of the cochlea the endosteal layer is, as a rule, missing. It is also absent on many parts of the semicircular canals. In the latter area there are also other irregularities of the structure of the capsule. The periosteal layer penetrates into the core of the pyramid, invading the spaces between the rings of enchondral bone which surrounds the semicircular canals. Thus, in the area of the semicircular canals the core of the pyramid is built by enchondral and periosteal bone. At the convexity of the superior and that of the inferior semicircular canal the enchondral layer is frequently missing. Thus, the bony wall of the semicircular canals consists only of periosteal and endosteal bone. Finally, the wall of the internal auditory meatus consists of lamellar bone separated from the enchondral and periosteal layers by a wavy cement line.

LYMPHATIC LEUKEMIA

C. R., a 62 year old woman, had suffered from chronic lymphatic leukemia for many years and complained of frequent bleeding from gums and vomiting. On Dec. 23, 1939 her hemograms showed 5.4 Gm. of hemoglobin per hundred cubic centimeters, 1,830,000 erythrocytes and 16,000 leukocytes, of which 97 per cent were lymphocytes, 2 per cent neutrophils and 1 per cent eosinophils. On December 25 she died.

At autopsy were found: leukosarcoma; leukemic infiltration of the dura and of the viscera; extramedullary erythropoietic islands in the spleen; fatty degeneration of the liver; bilateral chronic fibrous pleuritis; abdominal adhesions; evidence of ancient removal of the gallbladder, the internal genitalia and the appendix; dilatation of the common and cystic ducts; atherosclerosis of the aorta and the coronary arteries; healed tubercles of the left lung; pitting edema of the lower extremities; splenic tumor, and cyst of the left kidney.

Macroscopic Examination of the Skull, the Brain and the Temporal Bones.—The calvarium was thicker than normal, the maximum thickness being 13 mm. The cut surface presented a dark red color. The dura was smooth and glistening. On the base of the skull were several light pinkish elevations of dura measuring up to 1 cm. in diameter. The mastoid air cells and the temporal squama were infiltrated by soft, grayish red leukemic marrow. The bony structures were otherwise normal. The venous sinuses were filled with partially clotted blood. The superficial cortical vessels of the brain were slightly congested. The various portions of the leptomeninges were elevated by collections of straw-colored fluid. The brain appeared normal. The bone marrow including that in the tip of the petrous pyramid was diffusely infiltrated by leukemic cells which morphologically closely resembled lymphocytes. Some sections showed numerous megakaryocytes, many with evidence of advanced degeneration. The granulocytes and the erythropoietic elements were markedly decreased.

In both temporal bones there was a large amount of bleeding in the marrow of the anterior surface of the pyramid and in the dome of the jugular bulb. The internal auditory meatus was extremely narrow owing to a hyperostosis of the superior wall. In the region of the cerebral opening of the right vestibular aqueduct there was a hyperostosis which hung over that area, producing a sulcus. This extended from the groove of the sigmoid sinus up to the opening of the vestibular aqueduct. It is possible that the sulcus had harbored a vein connecting the sigmoid sinus with the endolymphatic duct (fig. 1).

Microscopic Examination of the Right Temporal Bone.—Middle Ear: Only the mesial wall was preserved. The windows were normal. The mucous membrane contained leukemic infiltrations which were situated below the epithelium. They were not diffuse but rather formed nodules.

Membranous Inner Ear: The membranous cochlea was well preserved and showed atrophy of the spiral nerve which was particularly marked in the basilar portion. There was neither infiltration nor collection of pathologic fluids within the spaces of the cochlea. The sacculus, the utricle and the ampullae were normal.

The periosteum of the internal auditory meatus presented diffuse leukemic infiltration (fig. 2 *C*) which extended into the facial canal as well as into the canal for the nerve of the posterior ampulla. Never, however, did the infiltration invade the sensory organs of the inner ear. The nerves within the internal meatus were normal.

Bony Capsule of the Inner Ear: Both the endosteal and the enchondral layer were normal. Conspicuous was the great amount of cartilage within the enchondral layer.

Leukemic infiltrations were present in the majority of the marrow spaces within the periosteal layer. They were striking in those below the cochlea and on the anterior surface of the pyramid. At no point was there any leukemic infiltration of the dura, though only a thin layer of bone separated it from the marrow spaces (fig. 2 *D*). In the area of the sub-

Fig. 1.—Posterior surface of the petrous bone. *M* indicates the internal auditory meatus; *D*, dura within the internal auditory meatus; *Ac*, the cochlear aqueduct; *Av*, the vestibular aqueduct. Note the deep sulcus extending from the vestibular aqueduct toward the sigmoid sinus.

arcuate fossa and mesial from it where the dura penetrated deeply into the roof of the internal meatus the leukemic infiltration never reached the level of the posterior cranial fossa (fig. 2 *D*).

In some of the infiltrated marrow spaces the leukemic cells were so numerous that the space scarcely contained any other tissue. In others the leukemic cells occupied the center and were surrounded by thickened hyalinized periosteum. In still others only small numbers of leukemic cells were noted. These marrow spaces contained to a great extent hyalinized connective tissue and thrombosed capillaries. In these spaces the bony walls presented numerous Howship's lacunas, but there were no osteoclasts. There were other marrow spaces the walls of which were aplastic and occasionally presented a blue line of arrested deposition. Bone trabeculae within the marrow were often covered with osteoid substance, indicating that there was bony deposition within the infiltrated marrow spaces. Occasionally the leukemic marrow invaded a bone trabecula to such an extent that the trabecula became almost perforated, as seen in figure 2 *B*. This process is known as dissecting resorption of bone. Such findings are well known in osteitis deformans (Paget's disease), in which, however, the bone trabeculae are invaded by fibrous marrow instead of leukemic cells.

The mucous membrane of the pneumatic cells was edematous at many sites. Frequently it contained nodule-like infiltrations of leukemic cells which absorbed the bony walls of the pneumatic cells. Osteoclasts could not be observed.

Comment.—The findings in this case of leukemia may be summarized as follows: (1) replacement of the marrow of the tip of the petrous pyramid by leukemic cells; (2) replacement of the marrow spaces of the periosteal layer by leukemic cells; (3) hemorrhage in the marrow spaces at the anterior surface of the pyramid; (4) resorption of bone of the periosteal capsule; (5) hyperostosis in the internal auditory meatus; (6) hyperostosis at the cerebral opening of the right vestibular aqueduct; (7) nodule-like infiltration of the mucous membrane of the middle ear, of the periosteum of the internal auditory meatus and of the mucosa of the pneumatic cells by leukemic cells; (8) atrophy of the spiral nerve in the basilar portion of the cochlea.

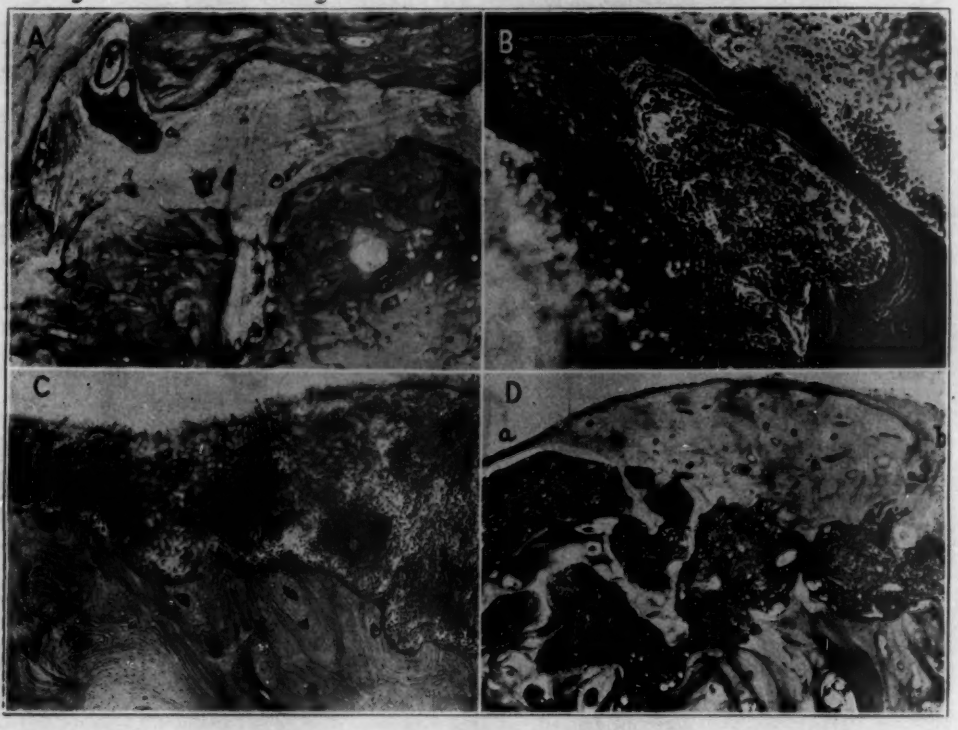

Fig. 2.—Leukemic infiltrations: *A* presents connective tissue originating in the dura of the posterior fossa and penetrating into the temporal bone. Within the connective tissue there are blood vessels (*a* and *b*) with dense perivascular leukemic infiltration.

B presents a bone trabecula within the periantral pneumatic cells. The leukemic infiltration of the trabecula has produced a large cavity the boundaries of which are covered by a fine seam of osteoid substance.

C presents the inferior wall of the internal auditory meatus the periosteum of which is markedly thickened by a leukemic infiltration which has eroded the bone by means of Howship's lacunas.

D presents leukemic infiltration within the enlarged marrow spaces of the periosteal layer of the bony capsule of the internal ear. In *a* the leukemic infiltration is separated from the dura of the middle fossa by a thin layer of bone; in *b* the leukemic infiltration invades the connective tissue but does not reach the dura.

Among these findings those concerning the bone are of importance. That the bone might be damaged by leukemic infiltrations of the marrow is well known.

Recently Apitz [1] studied this problem and found three particular osseous effects of leukemic infiltrations of bone: (1) focal lacunar absorption of normal bone, (2) damage of bone due to necrosis of the leukemic marrow and (3) leukemic osteoporosis consisting of general atrophy of bone, particularly of the trabeculae of the spongiosa.

In the temporal bone only lacunar resorption was observed, characterized by Howship's lacunas without osteoclasts, indicating absorption by osteoclasts at an earlier period of life. For this reason Howship's lacunas of the bony trabeculae are to be found in marrow spaces which are filled with hyaline connective tissue to a great extent, viz., in marrow spaces in which the leukemic infiltration was replaced by scar tissue. The finding of lacunar resorption of normal bone is interesting not only because it is rare in lymphatic leukemia but also because it is indicative of potential malignity of the leukemic cells.

The study of the case presented proves that only the periosteal layer of the bony capsule is involved in systemic diseases as far as the latter concern the skeleton. Many investigators have emphasized that only the periosteal layer participates in the vital processes of the skeleton under normal or under pathologic conditions. The mature enchondral layer and the endosteal layer do not present marked vital processes under normal conditions or under pathologic conditions unless a pathologic process originating in the periosteal layer or in the internal ear invades the enchondral layer. It is important to keep these facts in mind and to draw the following conclusions: If the pathologic process involves the enchondral layer only or chiefly, the disease began either in the fetal period or before the end of the second postfetal year. This is the period in which the enchondral layer has not yet reached its maturity. On the other hand, if the pathologic process involves the periosteal layer only, the disease might have begun in the fetal period as well as in any other period of life. Although these statements by no means involve unknown facts, they are often not considered as basic rules governing the pathologic changes of the bony capsule of the internal ear.

OSTEITIS FIBROSA OF VON RECKLINGHAUSEN

The case to be described was observed by Drs. K. M. Meyer, P. A. Rosi and A. B. Ragins for a long period. Dr. Meyer gave me permission to record the following observations on the temporal bones. Only a brief review of the clinical and postmortem observations is given. The details were published by Dr. Meyer and his associates.[2]

J. H., a 56 year old man, a Greek, was admitted to the Cook County Hospital on Oct. 8, 1937. He was perfectly well until April 1937, when he first noticed the gradual appearance of a slightly painful swelling of the proximal portion of the right little finger. On May 6 there occurred a fracture of the left humerus, and in August of the same year, a swelling of the right wrist. On his admission to the hospital, a firm tumor was felt to the right of the trachea and just above the sternoclavicular joint. There were enlargement and tenderness of the fifth digit of the right hand, of the distal portion of the right ulna, of the anterior surfaces of both tibias and of both clavicles. In view of these findings a clinical diagnosis of von Recklinghausen's generalized osteitis fibrosa due to adenoma of a parathyroid gland was made. There were renal calculi, a high serum calcium content, a low blood phosphorus content and a high blood phosphatase content. On October 28 Dr. Meyer removed a parathyroid tumor from the right side of the neck. Microscopic examination of the tumor revealed nonhornifying squamous cell

1. Apitz, K.: Ueber Knochenveränderungen bei Leukämie, Virchows Arch. f. path. Anat. **302**:301, 1938.

2. Meyer, K. A.; Rosi, P. A., and Ragins, A. B.: Carcinoma of the Parathyroid Gland, Surgery **6**:190, 1939.

carcinoma of the parathyroid gland. A biopsy was made of the swelling of the proximal phalanx of the right little finger. This swelling was found to be a giant cell tumor of the osteoclast type. Throughout the section there were numerous histiocytes laden with iron pigment.

In the early postoperative period there was a definite fall in the serum calcium with alleviation of the pains in the joints. Roentgen study of the bones showed definite recalcification of the fibrocystic areas. About ten months later, however, a distinct rise in the serum calcium was noted. The blood phosphorus was below its normal level while the blood phosphatase was slightly increased. This was interpreted as due to the persistence of parathyroid hyperactivity. The pains recurred. Recurrent decalcified areas were noted in some of the long bones. On Aug. 13, 1938 the right fifth finger was amputated. On examination the entire proximal phalanx was seen to be transformed into a globoid swelling. This when sectioned was soft and dark purplish red. The cortex of the bone was completely destroyed in some areas, while in others a thin shell was still discernible. The palmar structure of the finger in that area was pushed anteriorly but was not invaded by the tumor. A microscopic examination revealed a change similar to that which was observed in biopsy.

In the following months the osteitis fibrosa extended almost over the entire skeleton. The patient gradually became worse. On Oct. 23, 1940 the serum calcium was 13.66 mg. (serum calcium is normally 9 to 11 mg. per hundred cubic centimeters). On December 25 the temperature went up to 104.8 F. The patient perspired freely. There were moist rales over both lungs. The next day the patient died. A tentative diagnosis of bronchopneumonia was made.

At autopsy the essential observations were as follows: recurrent carcinoma of the parathyroid gland with metastases to the peritracheal, subclavian and perijugular lymph nodes, to the lungs and to the right kidney; diffuse osteitis fibrosa cystica involving practically all the bones of the body; multiple pathologic fractures of the right and the left femur, the left tibia and the left fibula; bilateral nephrolithiasis; chronic ascending bilateral pyelonephritis and left pyelonephrosis, chronic purulent cystitis; atrophy of the seminiferous tubules; brown atrophy and parenchymatous degeneration of the liver and the myocardium; cortical adenoma of the left adrenal gland; nephrocalcinosis.

The average thickness of the skull was 7 mm. The inner aspect was purple, gray and granular. The bone was somewhat flexible and sawed with ease. The leptomeninges were definitely edematous and slightly injected. The convolutions of the brain were slightly flattened. The lateral ventricles were slightly dilated. The vessels at the base were thin walled and smooth. The meninges covering the medulla oblongata and the cervical portion of the cord were brown-gray.

Microscopic Examination of the Temporal Bones.—The contents of the middle ear had been previously removed. There were no marked changes in the windows or in the promontory. Both sides presented a similar histologic appearance.

Membranous Inner Ear: The membranous inner ear was normal in its contour. In the vestibular and basilar portions of the cochlea there was definite atrophy of the spiral nerve and the spiral ganglion. In the other portions of the cochlea the nerve and the ganglions showed no abnormality. The bone of the modiolus was normal, and so was the pigmentation of the inner ear. There was atrophy of the stria vascularis in the basilar portion of the cochlea. Corti's organ was well preserved and consisted only of supporting cells, probably because of an artefact. The internal auditory meatus had a normal width, and its nerves were normal. The maculas and the cristae presented a very slight degree of atrophy. Both aqueducts were normal. The ductus and saccus endolymphaticus were changed into numerous narrow channels which frequently were without lumens.

Bony Capsule of the Inner Ear: A large portion of the capsule showed pathologic changes. Only the promontory presented a normal appearance although its inferior part or the boundary of the round windows showed pathologic involvement. In the upper boundary of the oval window the disease process had advanced almost to its cartilaginous framework. The fissura ante fenestram was substituted by the newly formed bone.

The most pronounced change was noted in the periosteal layer (fig. 3). The marrow spaces contained red or mixed marrow surrounded by a thickened

endosteum. Within the marrow there were bony trabeculae, some of which were normal and others pathologic. In the pathologic trabeculae the osteoclasts were either below the endosteum or within the vascular channels, indicating absorption

Fig. 3.—Cross section through the petrous pyramid: *M* indicates the middle cranial fossa; *Po*, the posterior cranial fossa; *Pn*, a pneumatic cell in the petrosal angle; *Mai*, the internal auditory meatus; *P*, the periosteal layer of the bony capsule of the internal ear presenting osteitis fibrosa; P_1, the normal periosteal layer separated from the affected periosteal layer in *x*; *E*, the enchondral layer of the bony capsule of the internal ear. The letter *a* indicates large marrow spaces containing fibrous marrow and invading the enchondral layer; *b*, newly formed bone within the enchondral layer; *c*, a sclerotic area; *d*, newly formed bone which almost comes in contact with the endosteum of the cochlea.

of bone from within and from without. The absorption from within or from the channels of the blood vessels was prevailing.

The bony septums between the pneumatic cells showed similar change. The pneumatic cells were either with or without a cellular fluid. The mucous membrane was edematous, and occasionally small edematous polypi were present. There was little absorption of bone originating in the mucous membrane; the latter presented rather osteogenetic activity by laying down new bone on the bony septums. There was, however, definite osteoclasis originating from the blood vessels within the bony septums. Within the channels of these blood vessels osteoclasts and fibrous tissue could be seen. By the activity of the osteoclasts the bony septums were destroyed in some places by dissecting absorption and in other places by absorption in the longitudinal axis of the bone trabecula. Thus, the channels of the blood vessels were gradually changed into marrow spaces filled with fibrous tissue.

These observations proved that the absorption of the periosteal layer did not originate in the endosteum of the marrow, in the mucous membrane of the pneumatic cells, in the dura covering the surface of the periosteal layer, in the connective tissue originating in the dura and invading the periosteal layer as in the subarcuate fossa or in the mucous membrane of the middle ear. The chief absorption set in within the channels of the blood vessels of the bone.

Comparison of Osteitis

Disease	Age of Occurrence	Sex	Hyperparathyroidism	Systemic Distribution	Spontaneous Fractures
Osteitis deformans (Paget's disease)	After 50	Mostly men affected	Not associated	Frequently observed	Seldom present
Osteitis fibrosa (von Recklinghausen's disease)	30 to 50	Mostly women affected	Associated	Always observed	Frequently present

The marrow spaces filled with fibrous tissue grew larger. They contained hyperemic capillaries, hemorrhages, occasionally some pigment and a loose connective tissue, some of which was rich in nuclei, giving the impression of sarcoma. The marrow spaces contained no cysts. Their fibrous tissue presented both osteoclastic and osteogenetic activity. The osteoclastic activity was due to osteoclasts which destroyed the old and the newly formed bone. The osteogenetic activity was due to metaplastic ossification of the fibrous tissue or to seams of osteoblasts. In either case most frequently bone of a primitive woven type was formed. It stained more with eosin when it contained a few cells and more with hematoxylin when it developed in the cellular connective tissue. The arrangement of the osteocytes was irregular, and the osteocytes did not present canaliculi. The newly formed primitive bone was later absorbed by the osteoclasts.

At the boundary of other marrow spaces osteoblasts were seen which laid down an osteoid substance and a more mature bone with strands of parallel fibers. With this formation of new bone the marrow space became narrower but it seldom disappeared entirely, since the new bone either ceased forming or became absorbed. In the first case a blue *Grenzscheide* (boundary line) appeared at the boundary of the marrow space. The fibrous tissue was replaced by fatty tissue. In the second case osteoclasts absorbed the newly formed bone in one area of a marrow space while in another of the same space the formation of new bone continued. It is noteworthy that in osteitis fibrosa absorption and deposition of bone do not follow each other in quick succession, nor are these processes located in close proximity as in osteitis deformans (Paget's disease). Therefore, in osteitis fibrosa irregular mosaic structures of bone are not as frequently observed as in osteitis deformans.

The transformation of the bone in the case presented was not limited to the periosteal layer. It extended into the enchondral layer and in some places even into the endosteal layer (fig. 3). The marrow spaces with fibrous tissue and the osteoclasts penetrated into these layers and simply destroyed the bone. In the newly formed marrow spaces new bone was laid down and was later absorbed. Thus the newly formed bone trabeculae replaced the enchondral layer and to a small extent the endosteal layer, but never perforated the endosteum and therefore never invaded the spaces of the inner ear.

It is noteworthy that the obliterated vascular channels within the enchondral layer frequently resisted the absorption more effectively than the bone and the chondroid tissue. Several years ago I emphasized that the channels of these blood vessels probably consist of precollagenous substance which stains deeply with hematoxylin. When the fibrous tissue originating in the periosteal layer invades the enchondral layer a channel of a blood vessel of the enchondral layer frequently lies within the fibrous tissue uncovered by bone. The channel might become thinner, but as a rule it does not disappear. Thus new bone is laid down around the old channel.

Comment.—In summary, there were (1) extensive transformation of the bony capsule of the internal ear and (2) atrophy of the spiral nerve in the basilar coil of the cochlea.

Deformans and Osteitis Fibrosa

Involvement of Skull	Sarcomatous Transformation	Cysts and Brown Tumors	Bony Cortex	Mosaic Structure	Deafness
Always present	Occasionally observed	Not present	Thickened, lamellated	Definite	Frequent
Occasionally present	Never observed	Present	Thinned	Not marked	Never present

These observations in the temporal bone are of particular interest as the changes were noted in a case of generalized osteitis fibrosa that was followed clinically for three years.

For a time the problems concerning the genesis of osteitis fibrosa (von Recklinghausen's disease) and osteitis deformans (Paget's disease) were considered solved. A number of clinicians and pathologists began to consider the two diseases as two different morbid entities characterized by the symptoms shown in the accompanying table. In recent years there has arisen again considerable diversity of opinion regarding the two diseases. Cases have been described which presented symptoms of both osteitis fibrosa and osteitis deformans. Husslein [3] and von Domarus [4] presented tumors of the parathyroid glands without hypercalcemia. Jaffe [5] and others noted instances of definite osteitis fibrosa with normal levels of calcium and phosphorus in the blood. Berblinger [6] recorded instances of osteitis deformans associated with

3. Husslein, J.: Zur Frage der Pagetschen und Recklinghausenschen Knochenerkrankung, Beitr. z. klin. Chir. **169**:276, 1939.

4. von Domarus, A.: Gibt es Uebergänge zwischen der Recklinghausenschen und der Pagetschen Krankheit? Deutsches Arch. f. klin. Med. **182**:611, 1938.

5. Jaffe, H. L.: Hyperparathyroidism and Its Relationship to Diseases of Bone, Bull. New York Acad. Med. **10**:539, 1934.

6. Berblinger, W.: Epithelkörperhyperplasie bei Osteodystrophia "deformans" (Paget) und bei abgeheilter Osteodystrophia fibrosa generalisata, Beitr. z. path. Anat. u. z. allg. Path. **94**:558, 1935.

tumor of the parathyroid glands. Michaelis [7] exhaustively discussed all these features.

The part played by the parathyroid glands is receiving much attention. In the past it was believed that tumors of the parathyroid glands produce a disturbance of the calcium-phosphorus balance, causing changes of the skeleton. However, experimental work has shown that disturbances of metabolism brought about by injections of solution of parathyroid or parathyroid extract, lead acetate, ammonium chloride or dextrose, prolonged anesthesia induced with ether or chloroform, a diet without vitamin D and poor in calcium, overdosage of viosterol or of vitamin A or poisoning with thallium acetate might produce essentially the changes of the bones which are characteristic of osteitis fibrosa. Therefore, osteitis fibrosa should not be considered as a result of a functional disturbance of the parathyroid glands. It is supposed to be rather the manifestation of a primary disturbance of metabolism consisting in relative acidosis combined with an increased amount of calcium in the blood. This disturbance of metabolism involves the skeleton by action of the parathyroid glands (Eger [8]).

One cannot reasonably hope that the microscopic examination of temporal bones would lead to a solution of these complicated problems. Nevertheless, the case presented might answer the question as to the manner in which a bone of particular structure responds to carcinoma of the parathyroid glands and to a definite disturbance of the calcium-phosphorus metabolism persisting up to the death of the patient.

The temporal bones have seldom been examined in instances of this type. Nager [9] presented a case observed by O. Mayer [10] and added 4 cases of his own which he considered as typical instances of osteitis fibrosa (von Recklinghausen). Among these 5 cases only 1 case presented all the symptoms of generalized osteitis fibrosa. The patient was a woman 37 years of age with multiple fractures, tumors of the parathyroid glands and calcium metastases in different organs. The pathologic changes were limited to the periosteal capsule of the internal ear. The blood vessels in the bony channels were dilated. There were fibrosis of the marrow and extensive osteoclasis and deposition of newly formed bone, laid down by osteoblasts. There were no cysts in the marrow. The enchondral layer of the capsule was normal. In 2 other cases of Nager the skeleton showed osteitis fibrosa but there were no tumors of the parathyroid glands. The temporal bones presented the same findings as did those in the first case. In one of these temporal bones the disease had invaded the enchondral layer of the capsule.

The case reported by Meller [11] concerned a child 2½ years of age who died from diffuse sclerosis of the brain. The case is not clear inasmuch as there were no clinical signs of osteitis fibrosa, although this disease was mentioned in the title of the paper.

Nager draws the following conclusions from his examinations: (*a*) The osteitis fibrosa of von Recklinghausen sets in exclusively in the periosteal layer. (*b*) The disease does not concern the enchondral layer unless the osteitis fibrosa is combined

7. Michaelis, L.: Histologischer Befund und Krankheitsbild, Klin. Wchnschr. **12**:1619, 1933.

8. Eger, W.: Die experimentelle Ostitis fibrosa generalisata und ihre Stellung zur renalen Rachitis, Klin. Wchnschr. **20**:353, 1941.

9. Nager, F. R.: Die pathologische Anatomie der Labyrinthkapsel und ihre klinische Bedeutung, Ztschr. f. Hals-, Nasen- u. Ohrenh. **34**:72, 1933.

10. Mayer, O.: Untersuchungen ueber die Otosklerose, Vienna, A. Hoelder, 1917.

11. Meller, H.: Zur Kenntnis der Osteodystrophia fibrosa des Schläfenbeins, Monatschr. f. Ohrenh. **71**:1293, 1937.

with otosclerosis. (*c*) The microscopic changes of the temporal bones are the same in osteitis deformans of Paget and in osteitis fibrosa of von Recklinghausen.

I am entirely in accord with the first conclusion. In addition I wish to emphasize that the osteitis deformans of Paget also begins in the periosteal layer. This I [12] emphasized many years ago, and it was recently confirmed by Tamari.[13]

I cannot agree with the second conclusion of Nager. In the case presented the enchondral layer was definitely involved without interference of otosclerosis. Therefore I consider that osteitis fibrosa might invade the enchondral layer as does osteitis deformans of Paget. Apparently it all depends on the duration of the disease. It might be stated that the transformation of the bony capsule of the internal ear in osteitis fibrosa does not occur as rapidly as in osteitis deformans.

The third conclusion of Nager is confirmed by the cases of O. Mayer,[10] Fischer,[14] Brunner and Grabscheid,[15] Wilson and Anson,[16] Tamari,[13] also by 5 cases of osteitis deformans of the temporal bone in which the bony capsule of the internal ear was microscopically examined by me.[17] However, the agreement concerns only the general aspect of these two diseases. In both osteitis fibrosa and osteitis deformans the bony capsule is transformed into trabecular bone containing the typical fiber marrow, hyperemic capillaries and occasionally pigment. I did not find, however, in the present case the perivascular infiltration which I have noted in the fibrous marrow of several cases of Paget's disease. The prevailing osteoclasis which is supposed to be significant of osteitis fibrosa of von Recklinghausen can scarcely be noted in the present case while in Paget's disease of the temporal bone the osteoclasis is frequently pronounced. The mosaic structures of the bony trabeculae were not marked in this case. However, they are also frequently not marked in Paget's disease of the temporal bone.

Summarizing I may state that in general the changes of the temporal bone are similar in osteitis deformans and osteitis fibrosa, although there are several differences in minute structure.

In osteitis deformans of Paget there are neither tumors of the parathyroid glands (except for the case of Berblinger) nor disturbances of the calcium-phosphorus balance. A question arises concerning the relationship between the changes of the temporal bone mentioned and the disturbances of the calcium-phosphorus balance in the case presented. There are two possible answers: Either the changes of the temporal bone have no relationship to the disturbance of the metabolism or they are related to the latter but do not present a specific response to it. In other words, the bony capsule responds to stimuli of different types with transformation into trabecular bone. The experimental research favors the second answer. Furthermore, inasmuch as the temporal bone presents essentially the same changes in renal rickets as in osteitis fibrosa and osteitis deformans (Chéridjian [18]), I conclude that the trans-

12. Brunner, H.: Ueber eine Fall von Paget'scher Krankheit, Montschr. f. Ohrenh. **56**:1, 1922.

13. Tamari, M.: Histopathologic Changes of the Temporal Bone in Paget's Disease, Ann. Otol., Rhin. & Laryng. **51**:170, 1942.

14. Fischer, J.: Studien zur pathologischen Anatomie des Schlaefenbeins, Monatschr. f. Ohrenh. **59**:586, 1925.

15. Brunner, H., and Grabscheid, E.: Zur Kenntnis der Osteitis deformans Paget der Schaedelbasis, Virchows Arch. f. path. Anat. **298**:194, 1932.

16. Wilson, J. G., and Anson, B. J.: Histologic Changes in the Temporal Bone in Osteitis Deformans (Paget's Disease), Arch. Otolaryng. **23**:57 (Jan.) 1936.

17. Brunner, H.: Die Beteiligung der knöchernen Innenohrkapsel bei Erkrankungen des Skelettes und des Knochenmarkes, Ztschr. f. Hals-, Nasen- u. Ohrenh. **29**:133, 1931. Brunner.[12] Brunner and Grabscheid.[15]

18. Chéridjian, Z.: Contribution à l'étude des répercussions morphologiques de l'ostéodystrophie rénale sur le rocher, Pract. oto-rhino-laryng. **4**:1, 1942.

formation of the bony capsule of the internal ear into trabecular bone with fibrous marrow is not always due to a disturbance of the calcium-phosphorus balance; it might also occur in diseases without disturbance of the calcium-phosphorus balance.

To appreciate better the pathology of otosclerosis the changes in the temporal bone in both osteitis fibrosa and osteitis deformans deserve much attention. Up to the present otosclerosis has not been entirely understood in spite of intensive research work. Nager and Meyer [19] have considered otosclerosis as a localized form of osteitis fibrosa. At the first glance there are several features which favor this hypothesis. Wittmaack [20] and Wilson and Anson [16] have refuted this concept. I am in full agreement with the opponents. There are many reasons for an opposing point of view; several of them have been emphasized by a number of investigators. The following points may be mentioned:

1. Otosclerosis is never a systemic disease.

2. In many instances otosclerosis is a localized disease of the bony capsule, involving particularlly the region of the windows. It frequently leads to ankylosis of the stapes. In so-called diffuse otosclerosis fusion of several otosclerotic foci occurs. Both osteitis fibrosa and osteitis deformans are from the beginning diffuse diseases of the bony capsule. They might extend to the windows of the internal ear but seldom lead to stapedial ankylosis. O. Mayer has emphasized that ankylosis of the stapes may occur in cases of osteitis deformans (Paget) with entirely normal mucosa of the middle ear. It is due either to a bony trabecula originating in the transformed bony frame of the oval window, partially substituting the stapes, or to metaplastic ossification of the annular ligament. The statement of Mayer is correct in general. However, ankylosis of the stapes was observed in 20 cases of osteitis fibrosa and osteitis deformans in but 2 instances. In these 2 instances the stapedial ankylosis was probably due to intercurrent otosclerosis. Although osteitis deformans, as a rule, does not produce stapedial ankylosis, it leads to deafness as frequently as does otosclerosis. However, the causation of the deafness is different in the two diseases.

3. The transformation of the bony capsule takes place more slowly in otosclerosis than in osteitis fibrosa and in osteitis deformans. The case presented shows that the speed of transformation in osteitis fibrosa is certainly not great. It took more than three years to transform a part of the bony capsule. However, in otosclerosis the speed of transformation is still less. Therefore, the intimate intermingling of resorption and deposition of bone so characteristic for osteitis fibrosa and osteitis deformans is, as a rule, missing in otosclerosis.

4. Otosclerosis, particularly the diffuse type, might lead occasionally to actual sclerosis of the bony capsule. This is seldom observed in either osteitis fibrosa or osteitis deformans of the temporal bone.

5. The newly formed bone due to otosclerosis frequently presents atrophic changes. This is particularly true on sites where it comes in contact with connective tissue, as in the fissura ante fenestram or at the endosteum of the internal ear. These atrophic changes are rare in osteitis deformans and osteitis fibrosa.

These facts certainly do not favor the concept of otosclerosis as a localized form of osteitis fibrosa. It must be conceded that up to the present otosclerosis is not a firmly established pathologic entity. Thus it is possible that some pathologic obser-

19. Nager, F. R., and Meyer, M.: Die Erkrankungen des Knochensystemes und ihre Erscheinungen an der Innenohrkapsel des Menschen, Berlin, S. Karger, 1932.

20. Wittmaack, K.: Betrachtungen zum Otoskleroseproblem, Hals-, Nasen- u. Ohrenarzt (Teil 1) **30**:337, 1939.

vations concerning otosclerosis which seem to be unquestionable at present, might in the future be considered as doubtful.

CONCLUSIONS

1. The enchondral layer of the bony capsule of the internal ear presents pathologic change if a disease began in the fetal period or in the infancy of the patient, unless a disease originating in the periosteal layer or in the internal ear has invaded the enchondral layer.

2. The periosteal layer presents pathologic change if a disease began either in the fetal period or at any time throughout adult life.

3. The periosteal layer of the bony capsule of the internal ear is the exclusive site from which both osteitis fibrosa (von Recklinghausen) and osteitis deformans (Paget) originate.

4. The enchondral and even the endosteal layer might be involved if the disease runs over a long period.

5. Neither disease passes over the boundaries set up by the endosteum of the internal ear.

6. In osteitis fibrosa and osteitis deformans the microscopic changes in the temporal bone are essentially the same.

7. The bony capsule of the internal ear might be transformed into trabecular bone with fibrous marrow from a nonspecific response to stimuli of a different type.

8. Otosclerosis cannot be considered as a localized form of osteitis fibrosa.

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EFFECT OF THE SPHINCTERIC ACTION OF THE LARYNX ON INTRA-ABDOMINAL PRESSURE

AND ON THE MUSCULAR ACTION OF THE PECTORAL GIRDLE

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I

A STUDY OF THE LITERATURE ESTABLISHES THE FACT THAT THE LARYNX CAN UNDERGO A SPHINCTERIC CLOSURE

The function of the larynx is by no means limited to its role in the production of voice. Perhaps of even greater importance, at least from the standpoint of the physiologist, is its action as a sphincter functionally separating the upper from the lower respiratory passages. This action was recognized long ago. Wyllie,[1] for instance, in 1866 pointed out that closure of the glottis takes place as a sphincteric phenomenon under three circumstances, i. e., during phonation, during deglutition and when air is compressed in the lungs and trachea whether voluntarily or involuntarily, as before each act of coughing. Wyllie apparently was not the first to make such observations, for he referred to "Mayo's Physiology," which I have been unable to trace. In addition, Czermak[2] five years before him described approximation of the true and the false vocal cords, resulting in hermetic closure of the glottis. Marshall[3] in 1868 said, "The glottis besides being the organ of voice acts as a safety valve preventing the intrusion of foreign bodies . . . into the windpipe. . . ."

Negus[4a, b] exhaustively described this sphincteric closure and its purposes and quoted Darwin[5] much to the same point. Starling[6a] pointed out that "the false cords function in a similar manner to the mitral valve of the heart," and Lauder-Brunton and Cash[7] wrote that "closure of the glottis plays a most important part in all expulsive acts such as coughing, sneezing, vomiting or defecation; or in those muscular actions where it is necessary to have the thorax fixed. . . ."

From the Bronchoscopic Clinic of the Cedars of Lebanon Hospital, Los Angeles.

Thesis submitted to the Faculty of the Graduate School of Medicine of the University of Pennsylvania toward the requirements for the degree of Doctor of Medical Science (D.Sc. [Med.]) for graduate work in otolaryngology.

1. Wyllie, J.: Observations on the Physiology of the Larynx, Edinburgh M. J. **12**:214, 1866.

2. Czermak, J. N.: Treatise on the Laryngoscope, translated by G. D. Gibb, London, New Sydenham Society, 1861, vol. 11.

3. Marshall, J.: Outlines of Physiology, Philadelphia, Henry C. Lea, 1868.

4. Negus, V. E.: (*a*) The Mechanism of the Larynx, London, William Heinemann, Ltd., 1929; (*b*) Evolution of the Speech Organs of Man, Arch. Otolaryng. **28**:313 (Sept.) 1938. (*c*) Negus,[4a] p. 236. (*d*) Negus,[4a] p. 246. (*e*) Negus,[4a] p. 252.

5. Darwin, C.: The Expression of the Emotions, New York, D. Appleton and Company, 1873.

6. Starling, E. H.: (*a*) Principles of Physiology, ed. 7, Philadelphia, Lea & Febiger, 1936; (*b*) ibid., p. 288.

7. Lauder-Brunton, T., and Cash, T.: The Valvular Action of the Larynx, J. Anat. & Physiol. **17**:363, 1883.

Sonnenschein, discussing a paper by Mullin,[8] described closure of the false and the true cords of the larynx to prevent invasion of foreign matter and in coughing. Jackson and Jackson[9] stressed that the function of the ventricular bands is to assist in the closure of the airway, the purposes of such closure being "protective, tussive and expectorative." Freedman[10] described the larynx as a "valve situated at the entrance of the pulmonary system with the primitive function of preventing anything but air from entering the lungs. . . ."

Stuart[11] from direct observation accurately described the gross mechanism of the closure of the sphincter of the larynx, including the levels of the aryepiglottic folds, the false and the true cords, and pointed out the sphincteric nature of this closure, adding that the true cords prevented the exit of air but not its entrance, whereas the reverse is true of the false cords. He also quoted Eberth,[12] who in 1868 observed tight closure of the larynx by the aryepiglottic folds. Lister[13] also reported closure of the false vocal cords as a sphincter during coughing and closure at a higher level (the aryepiglottic folds).

References to this subject are similarly found in many standard texts such as that of Thomson.[14]

My own article[15] added certain details to the previously known facts. It consisted of a description of the appearance of the sphincter as it opens and closes, together with photographs of this action and diagrams to indicate the muscular action involved.

It can therefore be accepted as well established that the larynx is capable of undergoing a sphincteric closure and that this consists of three tiers, the aryepiglottic folds, the false vocal cords and the true vocal cords, the latter two preventing respectively the exit of air from and the entrance of air into the lower respiratory passages.

A discussion of the far reaching effects of this action on man's ability to increase intra-abdominal and intrathoracic pressure for any purpose and on the efficiency of the action of the pectoral muscle girdle constitutes the purpose of this presentation.

II

EXCERPTS FROM THE LITERATURE INDICATE THAT THE MECHANICAL EFFECTS OF THE ACTION OF THE LARYNGEAL SPHINCTER ARE DUE TO VALVELIKE CLOSURE RATHER THAN TO STRONG MUSCLE CLOSURE

Negus[16] pointed out that the primitive larynx was first developed (in lungfish) as a simple nonvalvular encircling muscular band like the anal or the pyloric

8. Mullin, W. V.: Physiology of the Larynx, Tr. Am. Laryng., Rhin. & Otol. Soc. **34**: 341, 1928.

9. Jackson, C., and Jackson, C. L.: Dysphonia Plicae Ventricularis, Arch. Otolaryng. **21**:157 (Feb.) 1935.

10. Freedman, A. O.: Diseases of the Ventricle of Morgagni, with Special Reference to Pyocele of Congenital Air Sac of Ventricle, Arch. Otolaryng. **28**:329 (Sept.) 1938.

11. Stuart, T. P. A.: On the Mechanism of the Closure of the Larynx, Proc. Roy. Soc., London **50**:323, 1891-1892.

12. Eberth, C. J.: Hochgradiger Defect der Epiglottis, Virchows Arch. f. path. Anat. **43**:135, 1868.

13. Lister, S. J.: Anaesthesia, in Holmes, T.: System of Surgery, Philadelphia, H. C. Lea's Son & Co., 1881.

14. Thomson, St. C.: Diseases of the Nose and Throat, ed. 4, New York, D. Appleton-Century Company, Inc., 1937, p. 573.

15. Pressman, J. J.: The Sphincter Action of the Larynx, Arch. Otolaryng. **33**:351 (March) 1941.

16. Negus, V. E.: (*a*) A Hitherto Undescribed Function of the Vocal Cord, J. Laryng. & Otol. **39**:1, 1924; (*b*) footnote 4 *a* and *b*.

sphincter. In the course of evolution, however, it became a progressively more complicated structure and now in man depends for its sphincteric effect not so much on its muscular strength as on its valvelike qualities. This has been known for a long time and has been more or less proved experimentally by several investigators.

Thus Marshall [8] in 1868 spoke of the glottis as a "safety-valve." Negus [4c] discussed the shelflike inferior thyroarytenoid fold with its flat surface uppermost and capable of preventing the entrance of air in a valvular manner far more efficiently than could the sphincteric muscle action alone. The free ventricular folds or "exit-valves" prevent the escape of air from the trachea, and in a closed position they seal together all the more tightly when the pressure from below is increased. He summed up his observations on the point by indicating that the larynx can prevent the ingress or the egress of air by muscular sphincteric action or by a mechanism in which the true vocal cords or the false vocal cords act as a valve. The true cords act as a valve preventing the ingress of air, and the false cords, the egress. The purely muscular sphincteric effect which is present is not nearly as powerful as these valvular actions.

Long before Negus, however, in 1883, Lauder-Brunton and Cash [7] made important studies on the valvular action of the larynx. They demonstrated on the larynxes of cadavers by a brilliant suture fixation method that the closed ventricular bands were able by valvular effect alone and without muscular tension to withstand and offer complete resistance to a head of pressure from below equaling 30 mm. of mercury. The closed ventricular bands, however, offered no resistance to pressure from above, thereby further proving that their effect was not due to sphincteric muscle strength, for if it were, resistance would be offered equally to both ingress and egress; their shape and position enabled them to act as one way valves preventing the escape of air from below but not its entrance from above. The true vocal cords, on the other hand, resisted pressure from above equal to 140 mm. of mercury (which was not increased by simultaneous closure of the false vocal cords) but offered no resistance to the pressure of air from below. Thus they also acted as one way valves preventing not the egress of air, as in the case of the false cords, but the ingress of air. These observations are still accepted as the true facts.

Wyllie [1] in 1866 published one of the earliest and most complete accounts of this action and described the anatomic basis on which such valvular action depends. He stated that if a vertical section were made through the larynx so as to divide both the false and the true cords transversely, it would be found that when these are approximated the cavity of the larynx above the false cords as well as that of the trachea below the true cords is wedged shaped. When the true cords are thus in apposition, their upper surfaces present a broad flattened plane, slightly hollowed out on each side. On their tracheal aspect they are observed to fall away obliquely outward and downward, thus leaving an angle of considerable size which forms the margin of each vocal cord. The same obliquity is noted above the false vocal cords.

From this description it is obvious that Wyllie recognized the fact that pressure from below could force open the true cords but that this same pressure would wedge the false cords together even more tightly, with a similar situation existing but with the direction of pressures reversed, in the instance of the true cords. He clearly stated that the strength of the muscle structures involved in these closures is entirely inadequate to resist the enormous power which air may be made to exert on the glottis either from above or from below. He pointed out that its ability to resist passage of air is valvular in nature and suited to control both the

entrance and the exit of air. Wyllie, like Lauder-Brunton and Cash, substantiated his observations by suture fixation experiments, the sutures being placed through the arytenoid cartilages of the larynxes of cadavers.

Jackson and Jackson [17] indicated the valvular nature of the larynx in quite another way. They pointed out that in instances of bilateral paralysis of the laryngeal adductor muscles prior to the development of a cadaveric position the adduction of these muscles results in a continued effective phonetic, protective and expectorative function, despite the fact that paralysis has left them in a much weakened state. This strongly indicates that valvular effects are involved.

Stuart [11] was able to confirm the findings of previous observers and concluded that the true vocal cords act as valves and when approximated prevent the entrance of air into the larynx by mechanical or valvular means rather than by strength of muscle action.

In the face of these writings, many of them very old, and in the absence of proof to the contrary or even of objections to such a theory in the literature, it becomes necessary to look on the action of the laryngeal sphincter as that of a two way valvular mechanism since it is far more effective than can be accounted for by strength of the muscles involved.

III

ORIGINAL EXPERIMENTS DEALING WITH THE EFFECT OF THE SPHINCTERIC ACTION OF THE LARYNX ON THE MOVEMENTS AND THE POSITION OF THE DIAPHRAGM

From evidence in the literature it can be determined that the position and movements of the diaphragm are of great importance in establishing and maintaining intra-abdominal pressure and that, largely because of this, a definite relation exists between intrathoracic (or more especially for the present purpose, intratracheal) and intra-abdominal pressure.

The literature of the subject contains a number of references to the fact that in order to elevate intra-abdominal and intrathoracic pressure to high levels it is necessary that the glottis be shut. I have already referred to a number of these articles and will refer to others later on, as specific points are introduced into the discussion. However, I have not been able to discover quantitative evidence on purely experimental grounds to indicate the direct relationship between ability to fix the diaphragm and the presence of a closed glottis. In other words, so far as I know, no one has yet measured the difference, if any, in the position of the diaphragm as intrathoracic or intra-abdominal pressures are increased under conditions that are similar save for the fact that in one instance the glottis is open and in the other it is closed. The observations herein recorded are intended to establish these details as part of the general picture of the role of the laryngeal sphincter in elevating intra-abdominal and intrathoracic pressure by conscious effort.

Subjects of Experiment.—From the large number of patients attendant on the bronchoscopic clinic of the Cedars of Lebanon Hospital in Los Angeles a group of 16 volunteers was selected as suitable and used for the main portion of this work. From this group of volunteers 5 were used for the present portion of the experimental work. (The entire group, including these 5, was used in other experiments to be discussed.) One of the 5 patients had minor bronchiectasis, 1 had an inactive small pulmonary abscess and the other 3 had carcinoma of the larynx. Of the latter 3 patients, 2 were selected because their larynxes were completely occluded by large neoplasms and edema and the third because, while he still wore a tracheotomy tube, his larynx after roentgen treatment appeared entirely normal and the sphincteric action took place normally. Decannulation was simply postponed pending

17. Jackson, C., and Jackson, C. L.: Ventriculocordectomy: A New Operation for the Cure of Goitrous Paralytic Laryngeal Stenosis, Arch. Surg. **4**:257 (March) 1922.

completion of the experiment. Thus in these first 2 tracheotomized patients absolute control over the patency of the airway was available by the simple expedient of obstructing the tracheotomy tube, whereas the third patient, with the normal larynx, could be controlled only within the limits of the physiologic action of the larynx but still could be used as a control over the other 2 patients to demonstrate any deviations that might be expected from the presence of the tracheotomy tube per se, and for other observations as well.

Each of these 5 patients was extremely cooperative and intelligent and was selected because of willingness to permit any number of observations to be made. Each of the 2 patients not wearing tracheotomy tubes, in whose cases bronchoscopes were to be passed, had undergone bronchoscopy many times in the past, tolerated the bronchoscope extremely well without coughing and were accustomed to experimental work, having undergone experiments before for a different purpose. They were, indeed, ideal patients as subjects for experimental observations.

Experimental Procedure.—Each nontracheotomized patient was anesthetized locally by cocaine as for any bronchoscopic procedure. No preoperative medication was used. The patient was then placed in a recumbent position on an operating table under a 6 foot (approximately a 2 meter) biplane fluoroscope ordinarily used for the removal of foreign bodies. In order to avoid prejudice and to increase accuracy of observation, the fluoroscopic observations were made by a roentgenologist who was not informed as to the purpose of the experiment or its anticipated outcome. He was simply asked to fasten an identifying lead numeral with adhesive to the thoracic wall at the highest point reached by the diaphragm under abdominal straining and under various other conditions. The level of deepest inspiration was used as a base line, and this was marked with a different lead numeral. After the level of the diaphragm following each observation had been noted, repeated observations were made to establish it as the true level for each phase of the experiment.

Following the completion of the fluoroscopic examinations, a roentgenogram of the chest was taken at a distance of 6 feet with the various lead numbers maintained in situ by the adhesive strips, for the purpose of obtaining a permanent record. Measurements were then made on this x-ray plate, and the comparison between one diaphragmatic level and another could thus be readily determined.

The first phase of the experimental work involved the nontracheotomized patients and consisted in having each one, while under fluoroscopic observation as described, and under normal conditions, take as deep a breath as possible, the lowermost limit of the level of the diaphragm then being marked with a lead number. Following this, the subject was asked to strain as much as possible, simulating a forceful effort to urinate. Repeated observations were made after adequate rest to insure the accuracy of results. This portion of the experiment served to measure the height of the diaphragm resulting from maximum increases in intra-abdominal pressure with the sphincter of the larynx functioning normally.

A bronchoscope was then inserted to a level just below the glottis. The effect of this was to overcome any possibility of the sphincter of the larynx coming into play and to create a constant airway through it. Measurements taken under these conditions therefore represented the level of the diaphragm attained by maximum increases in intra-abdominal pressure without the action of the sphincter of the larynx. The difference in the two diaphragmatic levels (with and without the bronchoscope) therefore represented the effect of the sphincteric action of the larynx on the elevation of the diaphragm resulting from maximum efforts to increase intra-abdominal pressure.

An objection can be raised on the basis of a second variable, i. e., the presence of the bronchoscope itself, and for this reason the experiment was repeated with the 2 tracheotomized patients in whom there was pathologic occlusion of the larynx. By occluding the tracheotomy tube with a stilet a situation was created comparable to that of closure of the laryngeal sphincter since the outlet of the larynx itself was completely occluded by the pathologic alterations present. With the airway of the tracheotomy tube patent, the situation became analogous to that of the observations made with the bronchoscope in situ and therefore with no laryngeal sphincteric action.

Fortunately, a control over these 2 patients could be established by observation on the third tracheotomized patient, who had a normally functioning laryngeal sphincter. A sufficient amount of air passed around the tracheotomy tube so that within reason this patient simulated the normal but observations on him would bring to light any deviations due to the presence of a tracheotomy tube per se. The results were consistent, and therefore the tube itself was not a factor.

In no case were there pleural or diaphragmatic adhesions or a past history of pleurisy.

In the tracheotomized patients a second test was performed. Abdominal pressure was made by leaning heavily with both hands on the relaxed abdomen while the airway was open and while it was occluded. The purpose of this experiment was to determine whether closure of the airway prevented elevation of the diaphragm on increasing intra-abdominal pressure

passively, apart from any effort on the part of the patient. (The experiments with abdominal straining indicated the results on actively increasing intra-abdominal pressure.)

Results.—The results of these experiments are recorded in table 1.

With regard to patients 3 and 7 in this table the term "normal conditions" indicates that the airway was closed by virtue of the normal sphincteric action of the larynx. In patients 8 and 9, with pathologic stenosis of the larynx and a tracheotomy tube, the airway was closed by means of a stilet, and in patient 10, with a normal larynx and a tracheotomy tube, by a combination of both. Figures dealing with a persistently open airway appear in the columns headed "Bronchoscope in Situ" and "Tube Open." The latter term means that no occluding stilet is present in the tracheotomy tube and that air passes out through the neck without the interference of any sphincter or other occluding mechanism. In the former instance the presence of the bronchoscope in situ made any sphincteric action of the larynx impossible.

If one dares to base conclusions on averages, it is found that with the respiratory tract nonoccluded and in free communication with the outside world, as compared

TABLE 1.—*Elevation of Diaphragm from Lowest Respiratory Level With and Without a Closed Airway*

	Maximum Elevation, Cm.			
	On Active Attempts to Increase Intra-abdominal Pressure		On Applying External Abdominal Pressure	
Normal Subjects	Normal Conditions, Sphincter Physiologically Closed	Patent Bronchoscope in Situ		
3	3.5	5.5		
7	2.5	5.0		
Tracheotomized Subjects	Tracheotomy Tube Closed	Tracheotomy Tube Open	Tracheotomy Tube Closed	Tracheotomy Tube Open
8	1.0	2.0	1.0	2.5
9	2.0	4.5	3.0	4.0
10	1.5	4.0	2.5	4.0

with conditions in which occlusion is present either by virtue of the sphincteric action of the larynx or by occlusion of a tracheotomy tube, the maximum elevation of the diaphragm on actively attempting to elevate intra-abdominal pressure to the maximum averages 4.2 cm. in the former against 2.1 cm. in the latter—a relationship of exactly 2:1, the greater excursion occurring in the patients with nonoccluded airways. There were no exceptions to the general trend although the exact figure varied a little. In passive increase of intra-abdominal pressure by pressure of the hands on the abdomen, the ratio of maximum diaphragmatic elevations is as 2 is to 3 when the occluded airway is compared with the nonoccluded one.

It is to be noted that the same relationship is found regardless of whether or not the sphincter of the larynx is rendered nonfunctioning by the insertion of a bronchoscope, or whether or not there is a free escape of air through a tracheotomy tube below it. In other words, the presence of the bronchoscope itself did not affect the results because of some possible extraneous effects such as discomfort.

Conclusions.—The number of subjects studied is extremely limited, but it is not a simple matter even in a large bronchoscopic clinic to find more than a few who fully qualify for the intended purpose by (1) willingness to cooperate in such experiments, especially since this involves their undergoing further uncomfortable procedures yet to be described, (2) limitations of their pathologic condition that

make such experiments safe for them, (3) ability to tolerate bronchoscopic procedures with almost no emotional or untoward physical reactions, such as involuntary cough, abdominal muscular contraction or undue excitement, and (4) adequate intelligence to understand the problems involved, so that they can carry out the procedures required with minute understanding and in the face of discomfort.

What this group lacks in numbers it more than makes up in the ideal type of subject used. For this reason and because of the consistent results obtained, I feel justified in drawing certain fundamental conclusions from just this portion of the work alone. Added conclusions will be drawn from experiments yet to be described and their correlation cited.

It is obvious within the limits of the experiment that (*a*) by increasing intra-abdominal pressure as in abdominal straining for purposes such as defecation the diaphragm becomes elevated; (*b*) this elevation occurs whether the airway from the lungs is fully patent or completely occluded; (*c*) the degree of elevation of the diaphragm which takes place with the airway occluded either by the physiologic sphincteric action of the larynx or by the closure of a tracheotomy tube in situ is appreciably less than with the airway not occluded; (*d*) these observations apply to increases in abdominal pressure due to active contraction of the abdominal wall and to those due to the passive contraction of the abdominal wall resulting from outside compression.

IV

EFFECT OF THE SPHINCTERIC ACTION OF THE LARYNX ON INCREASES OF INTRA-ABDOMINAL AND INTRAPULMONARY PRESSURES AS DETERMINED FROM THE LITERATURE

The experiments just described demonstrated that changes in the position of the diaphragm take place as attempts are made to increase intra-abdominal pressure and that the elevation of the diaphragm is dependent, at least in degree, on whether the airway is patent or closed. From here one must go a step further in this same direction and indicate what relationship, if any, exists between the state of patency of the sphincter of the larynx and the ability of the human subject to increase intra-abdominal pressure. In other words, one wants to know how and to what degree these changes in diaphragmatic position can be correlated with ability to raise intra-abdominal pressure when the only variable is an open or a closed airway. In this manner may be demonstrated whether the ability to increase intra-abdominal pressure is or is not related to and dependent on the state of the laryngeal sphincter. It has already been shown how the position of the diaphragm is affected.

Langworthy, Kolb and Lewis [18] ascribed to observers as far back as Galen statements to the effect that increases in intra-abdominal or intrathoracic pressure, or both, are accompanied by a filling of the thorax with air, a fixing of the diaphragm at a low level and, with the stability thus produced, a squeezing down with the abdominal muscles.

It is my intention to point out that this action can take place with maximum effect only in the presence of a closed laryngeal sphincter or of some similar occluded mechanism substituted for the sphincter. This general observation in itself will be nothing new, but I propose to enlarge on it by submitting quantitative studies which I do not believe have been previously reported. It has already been shown by the experiments just described that stabilization and fixation of the diaphragm are dependent in part on a closed sphincter.

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18. Langworthy, O. R.; Kolb, L. C., and Lewis, L. G.: Physiology of Micturition, Baltimore, Williams & Wilkins Company, 1940.

Valsalva's experiment, which is well known and was cited by Lewis,[19] represents possibly the earliest effort intended to demonstrate these principles. It substitutes closure of the mouth and nares for closure of the laryngeal sphincter and in this manner prevents the escape of air from the thorax as intrathoracic and intra-abdominal pressures are forcibly increased by contraction of the thoracic and abdominal walls. This substitution of the closed mouth and nares for the closed laryngeal sphincter does not matter much, since the action of the laryngeal sphincter simply prevents the escape of air from the thorax at a slightly lower anatomic level. I think this will be adequately demonstrated in experiments that are to be described. Lewis pointed out from his own direct measurements that intra-abdominal and intrathoracic pressures increase tremendously during the performance of a Valsalva occlusion, so that in one experiment intrarectal pressure (as a measure of intra-abdominal pressure) reached a level of 94 mm. of mercury while intrathoracic pressure reached 87 mm.

Schaefer [20a] as early as 1900 stated that intratracheal air pressure equaled —0.1 mm. of mercury during quiet inspiration and increased to +0.13 mm. in expiration, but in Valsalva's experiment, with the mouth and the nose shut, thus occluding the airway, a forced expiratory effort may increase intrapulmonary pressure to as much as 100 mm. of mercury. Note that this is not very different from Lewis' [19] figure of 87 mm.

The increase of intrapulmonary pressure effected by occluding the airway (as, for instance, by closing the laryngeal sphincter) is by no means apparent solely in the presence of forced expiratory effort. It may be observed to a lesser degree under other circumstances. For instance, during systole of the heart, as a result of lessening of intrathoracic volume the pressure within the chest diminishes as compared with that during the diastolic phase.[20a] This relative diastolic increase in pressure does not occur, however, with an open larynx, but only when the breath is held and the laryngeal sphincter is closed.

Lewis [19] and Schaefer,[20a] among many others, therefore, recognized the role of an occlusion of the respiratory tract in increasing the pressures under discussion, but I do not find in their writings an exact reference to comparable results without this occlusion but with an otherwise similar effort.

Negus [4a] quoted Hobday, who in a personal communication to him presented in the most interesting way possible a practical observation to support the view of the close relation between intra-abdominal pressure and occlusion or nonocclusion of a respiratory outlet. If prolapse of the uterus of a cow occurs, it is sometimes necessary to perform tracheotomy before the organ can be replaced. Thus the "bearing down" is diminished in force, obviously because of the decreased ability to raise intra-abdominal pressure when the trachea is opened and the laryngeal sphincter is thereby rendered ineffective. It is obvious, therefore, that the raising of intra-abdominal pressure is controlled at least in part by the closure of the glottis and its sphincter valve or of some substituted similar mechanism.

Livingston [21] pointed out from the standpoint of practical observation that the effect of closing the laryngeal sphincter on intra-abdominal pressure can be observed in testing for ventral hernia. In changing from a supine to a sitting posture the patient contracts the diaphragm and the recti and associated muscles, thereby

19. Lewis, T.: Studies on the Relationship Between Respiration and Blood Pressure, J. Physiol. **37**:233, 1908.

20. Schaefer, E. A.: (*a*) Textbook of Physiology, London, Y. J. Pentland, 1900, vol. 2; (*b*) p. 279; (*c*) p. 346.

21. Livingston, E. F.: A Clinical Study of the Abdominal Cavity and Peritoneum, Am. J. Surg. **8**:1110, 1930.

increasing intra-abdominal pressure and causing the hernia to protrude, but this becomes most markedly manifest if a breath is taken first and the laryngeal sphincter then closed, indicating that such closure has a decided effect on the degree to which pressure within the abdomen can be increased.

Negus [4a] too described in great detail a closure of the larynx immediately prior to the expiratory phase of cough. The purpose of this is to permit, as the thorax contracts, accumulation of greater intratracheobronchial pressure, which is used to extrude foreign matter as the end result of the cough. That this procedure, dependent at least in part on laryngeal closure, also increases intra-abdominal pressure can be commonly observed in the extrusion of urine from a full bladder during cough, especially in persons in whom the sphincter of the bladder is paralyzed. It also becomes evident in the loss of feces, especially liquefied feces, under similar circumstances, and in the protrusion of inguinal and other hernias during the cough cycle.[21]

However, if a forced expiratory effort is made without closing the laryngeal sphincter and with no other difference from cough except this, the aforementioned phenomena do not occur. The conclusion to be drawn from these everyday observations is that laryngeal sphincteric closure is a necessary part of successful attempts to increase markedly pressure within the abdominal cavity.

In these attempts at raising intrathoracic and coincidentally intra-abdominal pressure the false vocal cords (or ventricular bands) play the more important role. I have previously described how the anatomic contour of these structures when they are approximated in the midline is better adapted to preventing the escape of air from the thorax than is that of the closed true vocal cords. I [15] have also demonstrated by still photographs and by motion pictures that laryngeal closure during cough is accompanied by closure of the false vocal cords and have graphically shown that this closure takes place not only in cough but in efforts to increase intra-abdominal pressure as in "bearing down" to empty the bowel or the bladder. Negus [4a] made the pertinent observation that in those species of mammals in which the false cords are not well developed, cough represents a much less powerful and effective effort than it does, for example, in man.

That the interrelationship existing between pressure increases within the abdomen, on one hand, and those in the thorax, on the other, is a close one and that in both cavities the increases of pressure are at least partially dependent on closure of the airway (as can be brought about for instance by closing the sphincter of the larynx or by the Valsalva experiment) was indirectly alluded to by Keith,[22] who pointed out important facts. Thus, he said, when the diaphragm contracts and the lower ribs become elevated a force is exerted on the abdominal viscera. But this force and the resulting increase in intra-abdominal pressure do not take place if the thoracic walls are sufficiently mobile. In that event the increase of pressure within the abdomen would in part be exerted upward and since elevation of the diaphragm is a passive act, not associated with contraction of any muscles, the resulting elevation due to pressure from below would force the rib cage outward, thus dissipating the energy intended to increase abdominal pressure, producing thoracic expansion rather than increased intra-abdominal pressure. This movement of the thoracic cage and forceful elevation of the diaphragm can take place more readily and more completely in the presence of an open laryngeal sphincter by virtue of the fact that the air within the thorax can be extruded, thus giving added space for the intrusion of the elevated diaphragm into the thorax. This presents

22. Keith, A.: The Nature of the Mammalian Diaphragm and Pleural Cavities, J. Anat. & Physiol. **39**:243, 1904-1905.

the less favorable situation for increasing abdominal pressure. But, with the thoracic cage fixed elevation of the diaphragm, according to Keith, takes place less readily, and it is perfectly logical that this should be the case. Such fixation of the thorax can be accomplished in no better way than by closing the laryngeal sphincter since air can then neither enter nor leave the thorax. With the quantity of air therein fixed, movement of the thoracic cage becomes limited. The reason for this is as follows: If under these circumstances the thorax were to contract without the possibility of air escaping from it, the effect would be to increase intrathoracic pressure and prevent elevation of the diaphragm. On the other hand if the thorax were to expand in the presence of a sphincter closed so that additional quantities of air could not enter, the increase in negative pressure in the intrapleural space would again have a decidedly limited effect on the thoracic expansion. Thus closure of the laryngeal sphincter valve tends to immobilize the thoracic cage regardless of whether a tendency exists to expand or to contract it, resulting in a state of thoracic stability which Keith described as being essential for the production of appreciable increases in intra-abdominal pressure.

Lauder-Brunton and Cash [7] recognized the validity of these observations. To quote them, "closure of the glottis plays a most important part in all expulsive acts such as coughing, sneezing, vomiting or defecation; or in those muscular actions where it is necessary to have the thorax fixed in order to enable the muscles attached to it to act with greater advantage or greater precision." Again, however, this is relatively a matter of conjecture on their part, since they present no actual measurements in the human subject with the larynx closed and then open, nor is the quantitative degree to which the larynx affects these physiologic activities indicated.

In the living human subject Jackson and Jackson [17] noted these effects in instances of bilateral paralysis of the recurrent laryngeal nerve. Thus, prior to the development of the cadaveric position, when the cords are more completely adducted, protective and expectorative functions are more effective. After the development of the cadaveric position, when this sphincteric effect is no longer present and a certain laryngeal patency adequate for the escape of air exists, these protective and expectorative effects (the latter being dependent on increases in intra-abdominal and intrathoracic pressures) are no longer equally effective. Thus the loss of the sphincteric effect produced by paralysis with the cords in the adducted position was accompanied by a loss of efficiency in these functions.

This inability to produce increased intrathoracic pressure in the face of loss of the sphincteric action of the larynx and the resulting diminution in the effectiveness of cough (as well as the loss of laryngeal protective action) were cited not only by Jackson but by Appelbaum,[23] who also observed it in a practical clinical way. In the face of heavy narcosis, when the laryngeal reflex is thereby abolished and the sphincteric effect no longer comes into play, not only does infected foreign matter more readily enter the tracheobronchial tree but it cannot be coughed out as efficiently, leading to production of pulmonary abscess.

V

ORIGINAL EXPERIMENTS TO INDICATE THE ROLE OF THE SPHINCTERIC ACTION OF THE LARYNX IN REGULATING INTRA-ABDOMINAL PRESSURE

Experiments Dealing with Voluntary Activity Attempts to Increase Intra-Abdominal Pressure.—To determine the part played by the sphincteric action of

23. Appelbaum, I. L.: The Respiratory Defense Mechanism, Am. J. M. Sc. **198**:356, 1939

the larynx in increasing intra-abdominal pressure, a series of experiments was conducted in which increases in pressure within the bladder were used as an indication of increases in the general intra-abdominal pressure.

The bladder was partially filled with water, and the rise of intravesicular pressure was measured during each phase of the experiment.

Resting pressure was established at zero, and patients were then asked to bear down utilizing their abdominal muscles as forcefully as possible in an effort which simulated that of forceful urination or defecation. Several readings were taken with a proper rest period after each, the maximum being reported in each case. Readings obtained on the cystometer represented the ability of the subject to elevate intra-abdominal pressure by forceful contraction of the abdominal walls with the diaphragm relatively fixed and the sphincter of the larynx closed. The former fact was established by observation under the fluoroscope, a phase which has already been discussed in a previous section, and the latter by glimpses with the direct laryngoscope, the use of which was a simple matter in patients so thoroughly accustomed to endoscopic procedures. The laryngoscope did not remain in situ during the actual taking of the reported readings, but the observation was made in an effort after the last intended reading had been taken simply to verify the fact that the sphincter of each particular person actually closed when abdominal straining was performed. No patient had to be ruled out because of failure of closure of the sphincter during the abdominal effort, although later on it was discovered that the sphincter of the larynx of patient 7 closed only once in many trials.

Six of the 10 patients so studied had either bronchiectasis or pulmonary abscesses, some with the latter type of lesion being now clinically well, and 1 had "cardiospasm." The other 3 were the tracheotomized patients described in the section on diaphragmatic studies. They were used with a specific purpose in mind, acting as controls.

After maximum ability to increase intravesicular pressure with the larynx functioning normally had been observed and recorded, the sphincter was rendered nonfunctioning by the passage of a bronchoscope between the vocal cords into the upper part of the trachea, which was well anesthetized with a 10 per cent solution of cocaine to prevent cough. The bronchoscopes used varied from a 5 mm. one for smaller women to a 7 mm. one for men. Thus a completely patent airway was established through the larynx. By having the subject repeat the abdominal squeezing and by recording the intravesicular pressure which resulted therefrom, the ability of each to increase intra-abdominal pressure without the action of the sphincter of the larynx was determined. The difference observed between intravesicular pressures on straining under normal conditions and with the sphincter nonfunctioning because of the unobstructed airway represents the quantitative effect of the sphincteric action of the larynx on the subject's ability to increase intra-abdominal pressure.

Certain controls were deemed necessary to determine whether the results obtained might be due to the presence of the bronchoscope apart from its preventing closure of the sphincter and establishing a patent airway. As in the experiments on diaphragmatic excursion, it is not likely to be universally agreed that the results obtained were due simply to differences in patency of the airway through the larynx; they might have been due simply to discomfort from the presence of the instrument even though in each instance the patient was well accustomed to the presence of a bronchoscope, having been treated bronchoscopically semiweekly over periods of several years. For this reason patients wearing tracheotomy tubes and well conditioned to the presence of the tubes were used in a limited sense as controls. Two of the 3 tracheotomized patients had, as described in the section on the diaphragm, larynxes completely occluded by the pathologic processes present so that straining with a patent and with a nonpatent airway could be obtained at will by having the tracheotomy tube open or occluded with a stilet respectively. It is acknowledged that this does not exactly qualify as a control for the studies of the action of the sphincter, but it did establish a state of affairs in which the effect of abdominal straining could be determined with a patent airway, on one hand, and a closed airway, on the other, with other variables reduced to a minimum. Since, however, the sphincter presumably accomplishes no more than a mechanical valvular closure such as that resulting from the occlusion of the tracheotomy tube in the presence of a pathologically closed larynx, experiments with these tracheotomized patients do serve as a control of limited value so far as the action of the laryngeal sphincter is concerned. Other control experiments have been made as well, and these will be considered later on.

The third tracheotomized patient (no. 10) was about to be decannulated. He had had a very small carcinoma of the anterior third of one vocal cord which had been treated by roentgen radiation. According to the routine of the department preliminary tracheotomy had been done some months previously, prior to beginning treatment. At the time of the experiment, the larynx appeared to be entirely normal, both the disease and the roentgen ray reaction having completely subsided. With the tracheotomy tube occluded in the presence of the normal larynx,

normal conditions of sphincteric action of the organ prevailed. With the tracheotomy tube nonoccluded, the escaping air was shunted out of the trachea so that the physiologic effects of the action of the sphincter were no longer present. Under this circumstance the experiment simulated those with the bronchoscope in situ, air being piped from the trachea to the outside world without being trapped by the laryngeal sphincter. It served to control these observations by doing away with the possible complicating effects of the presence of the bronchoscope apart from its action in piping air past the sphincter.

The experiments in this portion of the work were carried out on 10 volunteers, all patients in the bronchoscopic clinic of the Cedars of Lebanon Hospital, in Los Angeles. This includes the group of 5 who were used for the diaphragmatic observations, the experiments about to be reported having been performed on them in addition to the previous studies. Not all studies were performed on each patient simply because at the outset of the experiments they were not intended to include all observations subsequently made, so that the data are somewhat less complete as regards those studied first.

Each patient was locally anesthetized for the insertion of the bronchoscope and for urethral catheterization as well and placed in the reclining position on the operating table. An appropriately large-sized urethral catheter was passed into the bladder and fixed in situ with adhesive strips. The bladder was then slowly filled with isotonic solution of sodium chloride, 50 cc. at a time, until some discomfort resulted. The total amount varied between 200 and 350 cc. After each fractional instillation a sufficient time was allowed for any possible vesicular contractions to cease. When the bladder was filled, the lower level of the manometer was attached to the upright so that it rested at the level of the symphysis pubis, which was considered to be zero level. The catheter was then attached to the manometer. As I have already indicated, in 5 cases the mercury manometer was used, and in 5 others the water manometer. For the sake of establishing a control, 2 patients agreed to have observations made on them with both the water and the mercury manometer. Thus this portion of the experiment was performed twelve times in all, on 10 patients.

It might be added that following the removal of the bronchoscope a control reading under normal conditions was taken to be certain that any possible lowering of pressure within the bladder with the bronchoscope in situ had not been due to fatigue. If no undue fatigue was present, this control reading should approach closely to the original findings without the bronchoscope in situ. No undue fatigue was experienced, and no experiments were discarded for this reason.

Patients were not instructed as to the depth of breath to be taken prior to the abdominal squeezing effort. However, at least one trial was made with the deepest breath possible in each instance. The maximum pressure reading obtained regardless of the state of filling of the lungs is the one quoted.

The results obtained are recorded in table 2.

From these observations certain guarded conclusions may be drawn. The presence of a bronchoscope within the trachea, allowing free passage of air and thus preventing the occluding action of the laryngeal sphincter, will not permit the development of as great a degree of intra-abdominal pressure as develops when the laryngeal sphincter is functioning. In 7 persons thus tested the average maximum intra-abdominal pressure represented a 31 per cent reduction from the maximum obtained under normal conditions, which was considered the 100 per cent level. The maximum individual loss was 47 per cent. The least loss was 12 per cent, in a small, weak woman who had appreciable difficulty in reaching her maximum figures in each instance. Her individual readings (these are not given in the tables) varied much more widely than the general run. It is interesting to note that when tested with the water manometer she presented not the least loss of intra-abdominal pressure in the absence of a functioning sphincter but the greatest, amounting to 59 per cent. (See figures in table 2 under "Bronchoscope in Situ.") In all likelihood this patient should be considered as not a suitable subject for the experiment. Motion pictures of her laryngeal sphincter in action taken later demonstrated that complete closure of the laryngeal sphincter took place only once in a great many trials. All the other patients, however, presented consistent closures.

The so-called "control" group, i. e., subjects wearing tracheotomy tubes, presented pressure readings which substantiated the results in experiments with

the bronchoscope. It was not realized until later that the entire tracheotomized group had been studied with the water manometer, but this should not have too great a bearing on the percentage comparisons. The loss in ability to increase intra-abdominal pressure when tracheal air was not intercepted by laryngeal occlusion but passed through the tracheotomy tube directly into the outside world averaged in the tracheotomized subjects 22 per cent. This is a lesser loss than in the experiments with the bronchoscope and it may be that apart from providing a passageway for air the presence of the bronchoscope by itself may have limited the ability of the patient to bear down as forcefully as he could have without it. In a sense, therefore, the controls do not entirely bear out the conclusions drawn as to percentage differences, the discrepancy amounting to the difference between

TABLE 2.—*Results of Experiments with Bronchoscope or Tracheotomy Tube and Voluntary Abdominal Squeezing*

Patient		Maximum Intravesicular Pressure During Abdominal Straining		Approximate Percentage Difference Between A and B
		A. Normal Conditions	B. Bronchoscope in Situ	
		Mercury Manometer Used. Pressure Is Expressed in Mm. of Mercury		
1. H. H.	Male—extensive bronchiectasis	44	28	36
2. L. M.	Female—very minor bronchiectasis	50	30	40
3. A. D.	Female—asymptomatic small pulmonary abscess *	94	70	25
4. G. A.	Male—extensive bronchiectasis	82	56	31
5.	Male—"cardiospasm"	82	60	27
6. P. S.	Male—healed abscess of the lung	102	54	47
7. F. M.	Female—minor bronchiectasis *	50	44	12
	Average	72	49	31
		Water Manometer Used. Pressure Is Expressed in Inches of Water		
3. A. D.	Female—minor bronchiectasis *	71 (180 cm.)	48 (122 cm.)	32
7. F. M.	Female—asymptomatic small pulmonary abscess *	44 (111.5 cm.)	18 (45.5 cm.)	59
		Tracheotomy Tube		
		A. Occluded	B. Patent	
8. E. H.	Male tracheotomized patient—larynx completely occluded by carcinoma and edema	54 (137 cm.)	47 (119 cm.)	11
9. S. W.	Male tracheotomized patient—larynx completely occluded by carcinoma and edema	48 (122 cm.)	31 (78.5 cm.)	35
10. J. M.	Male tracheotomized patient—normal larynx with tracheotomy tube	77 (195.5 cm.)	61 (155 cm.)	20
Average for 3 tracheotomized patients		60 (152.5 cm.)	46 (116.5 cm.)	22

* Patients 3 and 7 represent those tested with both the mercury and the water manometer.

22 and 31 per cent. The control figures show a considerable variance from case to case, from 11 per cent in one instance to 35 per cent in another. The best control case from the standpoint of otherwise similar conditions seems to be that of patient 10, who wore a tracheotomy tube in the presence of a normally functioning larynx. His difference observed through many trials in which consistent results were obtained was 20 per cent.

One may say, therefore, that when air is shunted from the trachea into the outside world through a bronchoscope or a tracheotomy tube an appreciable diminution in ability to increase intra-abdominal pressure occurs, which, depending on the experimental type of shunting used, varies on the average from about 20 to about 30 per cent but which in certain subjects may reach as high as 59 per cent (patient 7, water manometer) or as low as 11 per cent (patient 8), these extremes being infrequently observed and possibly due to variables which are unavoidable in clinical experiments of this type.

The still undetermined factor so far is of course the effect of the presence of the bronchoscope itself, and in any final analysis allowance must be made for this factor per se. Therefore, further control to determine the effect of the presence of the bronchoscope and to evaluate what proportion of the loss in ability to increase intra-abdominal pressure is due to escape of air through the bronchoscope and what proportion is due simply to the discomfort of the mechanical presence of the instrument must be made. Other control experiments have, therefore, been performed beyond those on tracheotomized patients. The experiments consisted of introducing a bronchoscope, having first established the normal increases in intra-abdominal pressure possible without it (which figures for each patient have just been cited in table 2), and then taking readings first with the bronchoscope patent and second with the escape of air through it prevented by the surgeon's placing his thumb over the proximal orifice. Patients were asked to compress their lips tightly around the bronchoscope so as to seal off any air that might escape between the bronchoscope and the rima glottidis. Unfortunately, the nostrils were not compressed, so that some air might have escaped, but it would be in minimum quantities since the bronchoscope used was relatively large for each patient and did not allow the escape of appreciable amounts of air around it. In any event, if air did escape while the finger occluded the proximal orifice, the abdominal pressures reached would be lessened, so that the effect of the presence of the bronchoscope itself would be exaggerated in the results and the role of the sphincteric action of the larynx diminished. This is erring on the safe side. However, the discrepancy due to nonocclusion of the nares probably does not amount to a great deal. With the bronchoscopic lumen thus occluded, the subject was again asked to bear down as forcefully as possible, several readings being taken.

TABLE 3.—*Results of Further Experiments with Bronchoscope in Situ and Voluntary Abdominal Straining*

	Maximum Intravesicular Pressure During Abdominal Straining, Mm. of Hg			Percentage Loss with Bronchoscope in Situ ‡		
	A	Bronchoscope in Situ				F
Patient *	Normal Conditions †	B Patent †	C Occluded	D Patent †	E Occluded	Due to Escape of Air (Loss of Sphincteric Function)
2	50	30	40	40	20	20
3	94	70	80	25	15	10
4	82	56	64	31	22	9
5	82	64	74	27	10	17
6	102	54	94	47	8	39
7	50	44	54	12	Gain of +8	No loss
Average for patients 2 to 7	77	53	67	30	12.5	17.5
Average without patient 7				34	15	19

* Occlusion experiments were not done on patient 1 because of the onset of paroxysms of coughing during each of several attempts.
† Columns A, B and D are from table 2.
‡ The percentages are approximate to the nearest whole numbers.

The results are given in table 3. The normal maximum figures given in the previous table are repeated for simplification of comparisons. A further description of the results outlined in this table follows:

Column A represents the maximum intravesicular pressure attainable under normal conditions of abdominal straining. This is considered to be the 100 per cent level.

Column B represents the maximum of several readings taken with the bronchoscope in the upper part of the trachea, and the difference between columns A and B therefore represents the loss of ability to increase intravesicular pressure in the presence of a patent bronchoscope in situ. This loss is expressed in percentages in column D.

Column C represents the maximum intravesicular pressure recorded on abdominal straining with the bronchoscope in situ but with its lumen occluded. The difference between these figures and those in column A therefore represents the loss due to the presence of the bronchoscope itself since the figures in both columns A and C were obtained with no air escaping from the trachea, in one instance because the laryngeal sphincter was closed and in the other because the bronchoscopic lumen was occluded. The sole variable was the mechanical presence of the instrument, and the effect of this would therefore be measured by the difference in readings between these two columns of figures (A and C). This difference (or loss in pressure due simply to the presence of the occluded bronchoscope with no air escaping) is expressed in percentage in column E, in which the figures were determined by subtracting the reading in column C from that in column A and figuring mathematically the percentage of the total maximum pressure attainable (column A) this loss represented. For example: In patient 2 the total maximum pressure obtained under normal resting conditions was 50 mm. of mercury. This represented 100 per cent. The pressure with the bronchoscope in situ but occluded was 40 mm. Subtracting 40 from 50 gave 10 mm. loss due to the presence of the bronchoscope with no air escaping in either case (50 mm.: 10 mm.:: 100%: x%; 50x equals 1,000; x equals 20). Therefore a 20 per cent loss, as indicated in column E, can be attributed to the presence of the bronchoscope over and beyond its action in allowing the escape of air through the laryngeal sphincter.

The ultimate purpose of the presentation of these figures is to determine the loss from maximum due to the escape of air when the sphincter is nonfunctioning. This loss represents therefore the effect of the functioning larynx on the ability to increase intra-abdominal pressure. By subtracting the loss due to the mechanical presence of the occluded bronchoscope (column E) from the total loss observed with the bronchoscope in situ and not occluded (column D) one can determine the difference representing the loss in ability to increase intra-abdominal pressure when air escapes from the trachea during the abdominal squeezing effort while discounting the effect of the instrument used to permit that escape. This, then, represents the true effect of the action of the laryngeal sphincter. The percentage figures thus obtained are given in column F.

With the percentage loss of patient 7 omitted, as I think it should be for reasons previously cited, one arrives at a final average loss of 19 per cent in ability to increase intra-abdominal (or at least intravesicular) pressure by a conscious purposeful maximum effort of abdominal squeezing when the subject is deprived of the action of the laryngeal sphincter. The individual variations within this group varied from a loss of only 9 per cent to a loss of 39 per cent. The final figures are therefore quite variable within this series of subjects, no doubt indicating that studies must be made with a considerably larger series before establishing any figure as average.

At least one fact is established, namely, that the action of the laryngeal sphincter does play an essential role in the physiology of voluntarily increasing intra-abdominal pressure, the extent of which, however, seems to be variable in different subjects.

It is in order at this point to examine the results of the series of experiments just described and to compare them with the percentage differences found in the control group wearing tracheotomy tubes. In the latter group, it may be recalled, 2 subjects each had a larynx completely occluded by the pathologic process and the third a normally functioning larynx. Figures were obtained for increases in intravesicular pressure from abdominal straining with air trapped in the trachea and with air allowed to escape, the difference representing the percentage of increase in effectiveness of such efforts made possible by occluding the tracheal airway. The percentage difference varied from 11 to 35 with an average of 22 for the 3 subjects, which, again owing to the small number of cases, cannot be interpreted as a final average.

This control series nevertheless presents a result which corroborates the findings by the bronchoscopic method. The average of that group (19 per cent) is sufficiently close to the average of this group (22 per cent) to lend added weight to each separate series of observations. If one simply states the original premise to be that trapping of air within the tracheobronchial tree (as is accomplished by the action of the laryngeal sphincter), regardless of the method employed, increases the ability to elevate intra-abdominal pressure by squeezing, then the two groups can be added together so that a series of 8 with complete data instead of 5 gives essentially an average quantitative figure of ± 20 per cent, which is more or less the same as the average for each group examined separately. The variations within this larger group range from 10 per cent at one extreme to 39 per cent at the other.

Since the size of the group could not be enlarged beyond this, further observations in the nature of controls were made within the group itself. It was felt that if adequate control experiments could be established and a sufficiently large number of observations made by different methods on even so small a series, any resulting figure that was consistent could be considered as approaching a value which would hold true regardless of the size of the group tested.

TABLE 4.—*Results of Experiment with Intubation Tube and Mouth Open*

Patient	Normal Maximum Pressure, Mm. of Hg	Maximum with Intubation Tube in Situ and Mouth Open, Mm. of Hg	Loss in Ability to Increase Intra-Abdominal Pressure Due to Presence of Tube, per Cent
3	94	28	70
4	82	52	36
6	102	48	54
7	50	46	8
Average loss due to presence of intubation tube			42
Average without patient 7			52

Additional experiments were therefore performed. In the first the action of the laryngeal sphincter was prevented by inserting into the larynx the largest size O'Dwyer intubation tube possible and having the subject bear down with the tube in situ. This permitted the air to escape through the larynx and simulated in the normal patient the situation which existed in the tracheotomized subjects at least as far as the presence of a persistently patent airway was concerned. It also simulated conditions existing in the patients undergoing the placement of a bronchoscope except that the intubation tube replaced the bronchoscope itself. Experiments were carried out as previously. Except for the presence of the intubation tube all other conditions of the experiment exactly simulated the conditions under which pressure within the bladder was measured with the bronchoscope in situ. The results obtained are given in table 4. Four patients were so tested.

The average loss, then, of 42 per cent (or 52 per cent without patient 7, who, as in previous experiments, presented a loss considerably below that of the others) represents the total loss in power of increasing intravesicular pressure by abdominal straining with an airway in the form of an intubation tube inserted through the larynx. Again, as in the experiment with the bronchoscope, it is important that one compensate as best one can for any loss that may be due to the discomfort of the tube or for loss due to any other effect of its mechanical presence except the provision of a lumen through the larynx. In the bronchoscopic experiments this was accomplished by occluding the end of the tube with the finger, a condition which cannot be duplicated with the intubation tube. However, a similar

state of affairs can be brought about by having the subject close his mouth and by squeezing his nostrils together. By this means any difference in pressure between the so-called normal, in which the sphincter occludes the tracheal airway, and the latter group of experiments, in which the intubation tube is in situ and the mouth and the nares closed to prevent the escape of air, will demonstrate the loss in pressure due to the mechanical presence of the intubation tube apart from the escape of air it affords. Then the difference between the total loss and the loss due to the mechanical presence of the tube will again, as in the case of the bronchoscopic experiments, give the loss due solely to escape of air through the sphincter.

Table 5 adds this new factor of prevention of the escape of air by occluding the upper respiratory tract by closing the mouth and the nares.

The percentages given in this table were determined in exactly the same way as those in table 4, the two tables being comparable in every respect. By subtracting from the total percentage loss (column 4) that portion not due to the escape of air (as determined by preventing the escape of air with the tube in situ) (column 5) one arrives at a percentage figure (column 6) representing the loss in

TABLE 5.—*Results of Experiment with Intubation Tube and Closure of the Mouth and the Nares to Prevent Escape of Air*

	Normal Maximum Pressure, Mm. of Hg	Maximum with Intubation Tube in Situ, Mm. of Hg		Loss with Tube in Situ and Mouth Open, per Cent	Loss with Tube in Situ and Mouth Closed, per Cent	Loss Due to Escape of Air (Loss of Sphincteric Function), per Cent
		Mouth Open	Mouth Closed			
Patient	(1)*	(2)*	(3)	(4)*	(5)	(6)
3	94	28	46	70	50	20
4	82	52	62	36	25	11
6	102	48	80	54	32	22
7	50	46	Not tested	8		
Average percentage of ability to increase intra-abdominal pressure dependent on action of laryngeal sphincter						18

* Columns 1, 2 and 4 are from table 4.

ability to increase intra-abdominal pressure due to the escape of air from respiratory passages through the tube. This loss, which under ordinary circumstances is prevented by the action of the laryngeal sphincter, represents the quantitative effect exerted by the latter in the phenomenon of voluntarily increasing this pressure by abdominal straining.

The results obtained in the small group of subjects represented by table 5 indicate that from the maximum possible under normal conditions there is a loss in ability to increase intra-abdominal pressure amounting to a little less than 18 per cent when the larynx is prevented from exerting its normal sphincteric function.

Summary of the Experiments to This Point.—Two groups of patients are involved, one, a group that was normal so far as the action of the laryngeal sphincter is concerned, and a second, tracheotomized group. The results of the three sets of experiments carried out have been mutually corroborative within reasonable limits. It has been found that no matter by what method the test was made the loss of air pressure from the lower respiratory tract diminished the average ability of each group to increase intra-abdominal pressure through voluntary abdominal straining by about 20 per cent (18 per cent minimum in one group to 22 per cent minimum in another). Under normal physiologic conditions the prevention of

the escape of air from the lower respiratory tract is a function of the laryngeal sphincter so that as far as the experiments have gone it is reasonable to conclude that the laryngeal sphincter valve enables such pressure to be increased by a mean rough average of 20 per cent over the pressure possible without it.

Unfortunately, the individual variations within each group are wide, varying from a minimum of about 10 per cent to a maximum of about 40 per cent.

In each group of experiments abdominal pressure was increased through a purposeful voluntary act on the part of the patient. This aspect of the situation brought many uncertainties into the general picture, and it was deemed advisable to make further observations, removing such uncertainties as far as possible. In other words, it was felt that the observations should be repeated by some means which would produce maximum increases in intra-abdominal pressure apart from voluntary purposeful acts on the part of the subjects studied. Experimental attempts to do this constitute the next phase of this work.

Experiments with Active Attempts to Increase Intra-Abdominal Pressure That Were Not Altogether Voluntary.—It will be recognized at once that attempts to increase intra-abdominal pressure by active rather than by passive means cannot be entirely independent of a volitional attempt on the part of the subjects and therefore are apt to be variable. However, the voluntary aspects can be reduced to a minimum and increases in intra-abdominal pressure nevertheless obtained by employing involuntary cough.[1]

The general proposition that abdominal pressure increases during cough is well known. Thus one sees during cough descent and further protrusion of an inguinal hernial sac due to the increases in intra-abdominal pressure which accompany the cough, protrusion of viscera from an abdominal wound during a surgical operation when the patient breaks into a paroxysm of coughing, and extrusion of contents of the bowel and the bladder during cough, especially when sphincters are paralyzed or when the bowel contents are particularly liquid.

Cough, primarily a matter of a temporary marked increase in intratracheobronchial pressure which accumulates below the sphincter of the larynx when that mechanism closes in the preliminary stage as the chest is compressed, is accompanied by an increase in intra-abdominal pressure, a relationship which is discussed in other parts of this presentation. After the intratracheal pressure has accumulated sufficiently, the laryngeal sphincter opens, and the sudden release of this pressure is the cough itself.[24]

The physiologic mechanism involved, so far as the present problem is concerned, is well reviewed by Brown and Archibald,[24b] who said:

> While cough may be a voluntary act, it is generally a purely reflex and unconditional response, depending upon local irritation in the air passages originating nerve impulses which are transmitted by afferent nerve impulses in the vagus nerve. The efferent portion of the reflex arc is completed through motor fibers innervating the intrinsic and accessory muscles of respiration. The excitation of these motor nerves results in a contraction of the muscles involved. The abdominal muscles contract, bringing about an increase in intra-abdominal pressure, which is transmitted through the semi-rigid diaphragm into the thoracic cage. The intercostal muscles, aided by the abdominal and lumbar groups, tend to elongate the bony frame-work

24. (*a*) Carlson, H. A.; Ballon, H. C.; Wilson, H. M., and Graham, E. A.: The Effect of Phrenicectomy upon Cough and Expectoration, J. Thoracic Surg. **2**:573, 1933. (*b*) Brown, A. L., and Archibald, E. W.: The Action of Cough upon Material in the Tracheo-Bronchial Tract: Experimental Study, Am. Rev. Tuberc. **16**:111, 1927. (*c*) Wiggers, C. J.: Physiology in Health and Disease, ed. 3, Philadelphia, Lea & Febiger, 1939. (*d*) Pressman, J. J.: The Action of the Larynx, J. Laryng. & Otol. **53**:672, 1938; (*e*) The Physiology of the Vocal Cords in Phonation and Respiration, Arch. Otolaryng. **35**:355 (March) 1942; (*f*) footnote 15. (*g*) Negus.[4a] (*h*) Schaefer.[20a] (*i*) Wyllie.[1]

of the thorax and thereby markedly decrease the average diameter of the latter. The accompanying contraction and fixation of the diaphragm further narrow the chest cavity, and complete the contraction of the boundaries of the thoracic cage, except for the upper segment represented by the neck. This is held rigid by the long muscles of the neck, leaving only the exit through the trachea unaccounted for. The sum total of contraction of all the muscles involved results in a sudden diminution of intrathoracic capacity, with a corresponding concentric rise in intrathoracic pressure. Hence the rise is uniform throughout the chest, and is uniformly exerted upon all parts of the lungs. An outlet for this increase in pressure is sought, and is found in the sudden escape of air through the trachea, larynx and buccal cavity. This sudden expulsion of air is recognized as the common manifestation of cough.

Thus the increase in intra-abdominal pressure resulting during cough is more or less independent of the will of the patient and its amount of increase is not solely due to a wilful compression of the abdominal muscles. It more or less automatically varies with the intensity of the cough. Cough itself may be voluntary or involuntary, so that, under the latter circumstances especially, any increase in intra-abdominal pressure which results may at least in a sense be said to be beyond the control of the subject. The abdominal compression is not in this instance a purposeful intended effort carried out at the request of the investigator or to a degree which may vary with the ability or the willingness of the subject to cooperate.

I have therefore used differences in bladder pressures obtained during involuntary cough cycles with and without the action of the laryngeal sphincter as a measure of the effect of the latter structure on the ability to increase intra-abdominal pressure, the latter observations being considered as evidence obtained apart from the voluntary cooperation of the patient.

With the patient reclining and with the bladder filled and attached to the manometer as before, the onset of a coughing paroxysm was established by the rapid introduction of a few cubic centimeters of monochlorophenol through a direct laryngoscope, which was immediately withdrawn. In most of the patients, especially those with bronchiectasis who had purulent secretions in their bronchial trees, the coughing was more or less prolonged and violent. During the paroxysms of coughing the pressure within the bladder was noted on the manometer and the maximum recorded. The remainder of the experiment as carried out simulated those previously done with voluntary abdominal straining.

As a second step the bronchoscope was passed into the upper part of the trachea, and the pressure within the bladder during the ensuing cough was again noted and the maximum recorded. In some patients the passage of the bronchoscope itself was sufficient to induce adequate paroxysms of coughing, and in others the instillation of additional quantities of monochlorophenol was required. This procedure therefore gave the height of bladder pressure with a patent airway through the sphincter, which airway was established by the bronchoscope.

As a third step the bronchoscopic lumen was occluded with the surgeon's thumb during the cough cycle and the bladder pressure recorded during this period of cough. In this instance it was of course necessary to remove the finger during the periodic inspiratory effort to allow the patient to get air. The thumb was replaced over the lumen during the expiratory phase of cough.

The readings thus obtained are given in table 6. In this instance I have included all the findings in one table instead of dividing them between two as previously.

Again as in previous tables the total loss due to the presence of the unoccluded bronchoscope is represented by the difference between column 1 and column 2. By occluding the bronchoscope and noting the pressure which then accumulates within the bladder during cough (column 3) one obtains the pressure in the presence of a temporarily closed airway. Therefore by subtracting the figure in

column 3 from that in column 1 a figure is obtained which represents the loss in abdominal pressure due to the undetermined factors incidental to the presence of the bronchoscope, apart from its action as a passageway for air through the sphincter. This loss figured in percentage of loss from the maximum (column 1) is listed in column 5.

The difference between the percentage loss due to the nonoccluded bronchoscope (column 4) and that due to the occluded one (column 5) gives the percentage loss incidental to the escape of air through the bronchoscope apart from the effect of the latter's mechanical presence. This is listed in column 6 as representing the percentage loss due solely to the diminution in thoracic pressure incidental to the escape of air from the trachea. This, as before, is interpreted as the percentage role played by laryngeal sphincter activity in elevating pressure within the abdomen by its occluding or air-trapping function during the cough cycle.

Results obtained by this method of testing, using more or less uncontrollable cough instead of voluntary straining, present an average loss of ability to increase

TABLE 6.—*Results of Experiments with the Bronchoscope and with Production of Involuntary Elevations of Intra-Abdominal Pressure (Cough)*

Patient	Maximum Pressure During Cough, Mm. of Hg			Percentage Loss		Percentage Loss Due to Escape of Air (Loss of Sphincteric Function)
	No Bronchoscope	Bronchoscope Patent	Bronchoscope Occluded	Bronchoscope Patent	Bronchoscope Occluded	
	(1)	(2)	(3)	(4)	(5)	(6)
1	90	52	64	42	29	13
2	64	48	60	25	6	19
3	85	47	63	45	26	19
4	78	42	60	47	23	24
5	Experiment not done *					
6	140	78	94	44	33	11
7	Experiment not completed †					
Average for patients 1 to 6						17+

* Patient 5 was no longer a patient in the clinic when these experiments were performed.
† The experiment was not completed since the patient became frightened on coughing against the pressure of the thumb occluding the bronchoscope.

intra-abdominal pressure due to the absence of sphincter function of 17 + per cent. This compares with a loss of 18 — per cent obtained in the intubation tube experiments and a loss of 19 per cent in the bronchoscopic experiments during active controlled abdominal straining (exclusive of the findings for patient 7; if these are included the loss in the latter group of experiments is 17.5 per cent). Note that in the experiments with cough the results are more consistent one with the other, ranging from a minimum of 11 per cent to a maximum of 24 per cent.

The aforementioned figures apply to those patients with normally functioning larynxes. I have performed similar occlusion experiments on the tracheotomized group, placing a stilet in the tube during the expiratory phase of cough. The loss in elevation of intra-abdominal pressure incidental to cough without a momentarily occluded airway as compared with an occluded airway averaged 29 per cent. This is considerably above the 17 per cent average loss in intra-abdominal pressure due to the escape of air during cough in the normal nontracheotomized subject, an inconsistency which may possibly be accounted for by the fact that with the pathologically occluded larynx (patients 9 and 10) and occlusion of the tube with a stilet a longer closure took place than in the instance of the normally functioning sphincter. This permitted accumulation of added pressure. In the experiments

with voluntary straining the tracheotomized group likewise presented a somewhat greater loss in ability to increase intra-abdominal pressure than did the others, although this was not nearly so marked as in these experiments with involuntary cough.

The results obtained on the tracheotomized group are given in table 7. (Note that the results are in inches of water instead of millimeters of mercury.)

One further series of control experiments was attempted, but the observations were not completed. An attempt was made to determine the differences in bladder pressure when my hands were pressing forcefully on the abdomen of the subject with and without a patent tracheal airway. This was to determine whether or not it was possible to raise intra-abdominal pressure by external means as effectively when the airway through the larynx was patent as when it was occluded. It was hoped to arrive at a figure which would give some evidence of the relative degree to which intra-abdominal pressure can be elevated when air is passively pushed out of the chest through elevation of the diaphragm from below, compared with

TABLE 7.—*Results of Experiments with Cough in Tracheotomized Patients*

Patient	Maximum Pressure, Inches of H_2O		Loss in Ability to Increase Intra-abdominal Pressure Due to Escape of Air, per Cent
	Cough; Tracheotomy Tube Occluded	Cough; Tracheotomy Tube Patent	
8	51 (129.5 cm.)	40 (101.5 cm.)	21
9	82 (208 cm.)	57 (144.5 cm.)	30
10	48 (122 cm.)	30 (76 cm.)	37
Average			29

TABLE 8.—*Experiment with External (Hand) Pressure*

Patient	Maximum Pressure		Decrease Due to Preventing Occlusion of the Airway
	Patent Airway	Occluded Airway	
8	20 mm. Hg	33 mm. Hg	39%
8 (tracheotomy tube)	11 in. (30 cm.) H_2O	21 in. (53 cm.) H_2O	50%

the increase possible when air was trapped by the laryngeal sphincter. These experiments were not completed, however, because, after making preliminary arrangements for carrying them out and actually doing two of them, I decided to change the method and substitute for hand pressure the use of an inflatable abdominal binder such as urologists used for intravenous pyelograms. A fixed measured amount of external pressure could then be used (by incorporating a blood pressure gage in the system) instead of the indefinite amount produced by pressure of the hands. Due to circumstances beyond my control it was not possible to carry out the latter group of observations. The results obtained with the 2 patients tested by crude hand pressure are as shown in table 8. One experiment was done with a water and the other with a mercury manometer.

These are essentially the same type of experiments as those carried out on diaphragmatic excursion (table 1, column 3) and when completed were intended to be correlated with them.

They cannot be considered significant because there was no way of knowing whether or not equal pressure was used in each instance.

This concludes a description of data obtained for the purpose of comparing the ability of an experimental human subject to voluntarily, and to more or less

involuntarily, increase intravesicular pressure in the presence of an intact, functioning laryngeal sphincter valve with that under circumstances in which the valve has been rendered nonfunctioning. For reasons cited from the literature the intravesicular pressures are interpreted as reflecting in a quantitative way the increases in the general intra-abdominal pressure.

Because of the difficulties involved in finding subjects suitable for experimental work of this type and willing to undergo the discomfort incidental to it, the series of observations reported is small, but a number of control observations have been made in each instance.

The findings reported are consistent within reasonable limits. Appreciable individual deviations from the average percentage figures are present, and this in so small a group lessens appreciably the value of the averages cited. Nevertheless a general pattern can be discerned.

VI

PSYCHOLOGIC PRINCIPLES INVOLVED IN THE ACTION OF THE SPHINCTER VALVE OF THE LARYNX AS A FACTOR IN THE ABILITY TO INCREASE INTRA-ABDOMINAL PRESSURE

I have pointed out from a study of the literature and from personal observations that the larynx can undergo a sphincter-like closure quite apart from its sound-producing function. This closure is effective by virtue of its valvelike qualities and comes into play under a variety of circumstances, one of which is the production of an elevation in intra-abdominal pressure. By the presentation of experimental evidence it has been demonstrated that this closure is not a purposeless accidental or coincidental phenomenon but plays an important role in a quantitative way, affecting the degree to which intra-abdominal pressure can be raised. Loss of this sphincteric function is accompanied by loss in the ability to raise pressure within the abdominal cavity. The diminution in this ability amounts to about 20 per cent with appreciable variation from one subject to the next. One subject (patient 10) presented results that seem especially significant. He had had a small carcinoma of the larynx which was treated by roentgen radiation so that at the time of the experiments he possessed a normally functioning larynx and at the same time wore a tracheotomy tube. By inserting a very short tube into the stoma and occluding it with a stilet one could simulate normal physiologic conditions. By simply removing the stilet and allowing air to escape through the tube one could nullify the effect of closure of the laryngeal sphincter. The resultant loss in the ability to increase intra-abdominal pressure amounted to 20 per cent.

The mechanism involved in such a loss seems rather obvious. Intra-abdominal pressure is elevated essentially by compression of the muscles of the abdominal wall. The force of this compression is exerted equally in all directions within the closed space of the abdominal cavity. All the walls of this cavity are thereby more or less fixed, with the exception of the superior wall consisting of the diaphragm, which is freely movable. Pressure exerted against this superior wall tends to force the diaphragm upward. If this dome is fixed, especially in a lowered position, thereby limiting the size of the abdominal cavity, increases in pressure exerted by contractions of the abdominal wall will tend to reach a maximum. If, on the other hand, the diaphragm remains mobile, then part of the pressure thus exerted is effective in elevating the diaphragm higher into the thorax so that the force of the muscular effort is exerted in part in enlarging the capacity of the abdomen and only in part in increasing the pressure within it.

In order for this upward displacement to take place, air must be forced out of the thoracic cavity. If it is not, upward movement of the diaphragm will be limited by the constantly increasing positive pressure above it and the diaphragm will become automatically fixed, a situation which is exactly what occurs when the laryngeal sphincter valve closes. Thus escape of air from the thorax is prevented, and the diaphragm becomes relatively fixed, furnishing a stable, firm superior wall to the abdominal cavity. This results in a maximum increase of pressure within the cavity instead of an increase in its capacity.

The correlation existing between closure of the laryngeal sphincter, trapping of air within the lungs and resultant fixation of the diaphragm can be demonstrated during efforts to increase intra-abdominal pressure. Observations outlined in part III have shown that diaphragmatic movements upward are greatly limited during abdominal straining when the sphincter action remains intact, but the upward movement increases appreciably when the valve is rendered nonfunctioning and there is a free airway permitting escape of air from the lungs. This added upward movement of the diaphragm by enlarging the capacity of the abdomen and failing to offer a firm superior wall to the confines of the abdominal space accounts for the 15 to 20 per cent loss in ability to increase intra-abdominal pressure. The trapping of thoracic air within the tracheobronchial tree to prevent upward excursion of the diaphragm is a function of the sphincter valve of the larynx and on the effective action of this organ therefore depends the ability to raise intra-abdominal pressure to its highest possible level.

One final point should, however, be made clear. The action of this laryngeal sphincter valve is not specific in any way. It is due solely to the fact that it occludes the airway at the level of the larynx, preventing the escape of air. Very likely any other means of occluding the trachea would be equally effective, and I have no doubt that in the absence of any valvular mechanism within the larynx a substitute for it could be found in such means as a tight occlusion of the lips and nostrils which might with equal efficiency prevent such escape of air from the respiratory passages. Nor do I consider the site of the occluding valve at the larynx particularly important. Except for the possible trifling effects of minor variations due to change in capacity of the respiratory passages, the valve could probably be located anywhere between the carina and the point of escape of air at the level of the mouth and the nares. Even below the carina, twin valves occluding the main stem bronchi should, theoretically at least, serve the purpose as well as the laryngeal sphincter valve. However, at the level of the larynx and within its confines nature found a convenient site and convenient means of effectively occluding the airway of mammals. One sees, then, that this sphincter, dating back in the phylogenetic scale to the Dipnoi or lungfish and originating as a circular sphincter muscle to keep sea water from rushing into their lungs, has remained throughout the various stages of evolution until now in mammals, including man, it has many duties to perform, not the least of which is its function in helping to make possible maximum elevations of pressure within the confines of the intra-abdominal space.

VII

THE ROLE OF THE SPHINCTER VALVE OF THE LARYNX IN THE MUSCULAR ACTION OF THE PECTORAL GIRDLE AS DETERMINED FROM THE LITERATURE

Up to this point I have considered the role played by the laryngeal sphincter valve during physiologic efforts to increase intra-abdominal pressure. Many other

functions have been ascribed to this valve, one of the more interesting being a part in increasing the efficiency of action of the pectoral group of muscles.[25]

Thus Negus [4d] said:

. . . It has been shown that almost invariably valvular (laryngeal) inferior folds are present in animals capable of independent use of the forelimbs. The two are so closely related that no explanation seems feasible except that the anatomical structure is designed to subserve the physiological function. . . . In animals, including man, when the forelimbs are pulled towards the ribs by the pectoralis major which arises from the ribs, it is necessary that the ribs be held in an immobile position in order that they may act as a fixed point of origin for the muscles. . . . The ribs may be held down by the action of muscles pulling on them from below, notably the rectus abdominis and other muscles of the anterior abdominal wall. But fixation solely by the action of muscles is an unnecessary expenditure of energy if some more economical mechanism can be evolved. And furthermore, muscular action would not give so steady a fixation as the method to be described. For these and other reasons I think it is beyond dispute that for efficient fixation of the thoracic wall it is necessary that air should be imprisoned in the thoracic cavity while the effort lasts, there being consequently a temporary cessation of respiration. To imprison the air the glottis must be closed. . . . The mechanism referred to is of course the inlet valve which in man consists of the thyroarytenoid folds (true vocal cords).

To this I would add that the ventricular folds or false vocal cords are essential too since, as I have previously pointed out and as Negus described elsewhere in his text, the true cords prevent the ingress of air but the false cords are necessary to prevent its egress. So far as fixation of the thorax by trapping of air is concerned, it is just as important to prevent the egress as the ingress of air. To continue with the quotation from Negus.[4e]

By means of the laryngoscope, the vocal cords of man are seen to shut with a snap on making an effort with the arms in an adducted position. . . . As the effort becomes greater, so the closure of the glottis is more forcible, and in some cases the ventricular bands approach each other and may touch along part of their length.

The converse, as cited by Thomson,[14] is that after total laryngectomy a patient finds that the absence of cords prevents him from fixing the chest for many actions. If a laborer is deprived of the larynx he cannot earn his living by manual labor.

Starling [6b] pointed out that as a means of holding the breath as in these pectoral girdle efforts "the false cords function in a similar manner to the mitral valve of the heart."

Negus [4a] included in his résumé of the subject the observation that during pectoral girdle action the closure of the laryngeal sphincter traps air in the thorax and fixes the thoracic cage, permitting greater efficiency of action by giving the pectoral muscles a fixed point of origin, and if fixation is in partial expiration, by depressing the rib cage, and thus lengthening the pectoral muscles, which further tends to increase their efficiency. The situation in this regard is summed up as follows:

If the laryngeal valve is closed at the moment when an animal wishes to use the forelimbs independently, no air can enter the lungs, and the thorax is to a certain degree prevented from expanding; the ribs therefore tend to remain at rest, and give a fixed point of origin to the pectoral muscles. The latter in their contraction exercise the full force of their pull on the humerus and move the forelimbs. If the ribs were not fixed, they would rise when the pectorals contracted, and the desired result of moving the forelimbs would not be so well attained. Some action of the abdominal muscles is required of course, to fix the ribs, but the mechanism described relieves them of much expenditure of energy.

Starling [6b] lent support to these views by his statement that animals which need the thorax to be fixed in order that they may climb or strike have well developed false vocal cords. Lauder-Brunton and Cash [7] wrote that closure of the glottis plays a most important part when it is necessary to have the thorax fixed in order

25. Negus (footnotes 4*a* and 16*a*).

to enable the muscles attached to it to act with greater advantage or greater precision. The false vocal cords are therefore more fully developed in those animals whose habits render thoracic fixation serviceable and vice versa. This actually seems to be the case, for one finds the ventricular bands absent in kangaroos, with feeble, underdeveloped forelimbs, and also in dogs and sheep, which do not use their forelimbs in any exaggerated way. However, in animals capable of the powerful acts of hugging, striking or climbing, in which the forelimbs play a predominant role, one finds strong, well developed, powerful ventricular bands, as in the lion, the tiger, the bear and the howling monkey. Negus [4b] included man in this group by pointing out his descent from an arboreal ancestry with its heritage of a valvular type of larynx.

Freedman [10] expressed these same views, namely, that in anthropoid apes and other mammals the pectoral muscles, in order to attain maximum efficiency for grasping, hugging, striking and climbing, must function in the presence of a fixed thoracic cage, which means a temporary cessation of respiration (as would necessarily occur in the presence of a closed laryngeal sphincter).

There are, therefore, in the literature a number of references to indicate a purpose served by the sphincter valve of the larynx in increasing the efficiency of the pectoral group of muscles. This premise seems reasonable by virtue of the theory of the muscular action involved and of studies of comparative anatomy.

VIII

ORIGINAL EXPERIMENTS ON THE ROLE OF THE SPHINCTER VALVE OF THE LARYNX IN THE ACTION OF THE PECTORAL MUSCLE GIRDLE

I have always assumed that the theory just described was correct and have in certain previous writings subscribed to it without raising any question as to its validity. Experiments have been carried out which were intended to permit study of the phenomenon in a quantitative way, rather than to disprove it. As a result of them, however, I have not been able to observe any significant quantitative loss of pectoral girdle efficiency in the absence of a functioning laryngeal sphincter valve.

The experiment consisted of having each of a number of human subjects adduct the arms across the chest in a manner simulating hugging, a movement which had been observed at my request by qualified orthopedic physicians and accepted as a typical function of the pectoral muscle girdle. The subject grasped in each hand a handle removed from a weight-pulling exercising apparatus. Each handle, by a rope and pulley arrangement, was attached to a separate spring scale, the subject pulling against the resistance of the two scales. Thus the amounts of pull in pounds of the right and left arms separately could be observed. The subjects were reclining on their backs, and the scales were attached by an extension bar to the sides of the operating table. The elbows were flexed at about a right angle and the wrists were maintained in a straight line with the forearm, without flexion or extension. All movement of the arms was at the shoulder joint. As the closed fists passed across the midline of the body they were moved slightly up or down, so as to avoid each other.

In order not to complicate the figures the sum total of the pull in the right and left arms is given, since no significant difference in results has been observed by considering each arm separately.

Readings of the strength of muscle pull were taken with the patient reclining under normal conditions, with no instrumentation of any sort and with no directions as to whether to hold the breath or whether to make the effort during inspiration or expiration; they were simply to exert the maximum possible pull. All patients, however, without directions to do so, held their breath during the effort.

In the normal subjects the experiment was then repeated with a nonoccluded bronchoscope in the upper part of the trachea and with the surgeon's thumb over the proximal orifice of the tube, occluding it.

In the tracheotomized patients the pull was made with the tube patent and then with the stilet occluding it.

Each observation on any subject was repeated a number of times to be certain that the maximum pull of which the subject was capable had been attained.

The results of the first of these experiments are recorded in table 9. The figures indicate that while in some of the 8 subjects there was a drop in pectoral muscle strength when a bronchoscope in the upper part of the trachea prevented closure of the laryngeal sphincter valve, I was able to observe but one increase above this lower figure when the lumen of the instrument was occluded. The interpretation to be placed on this is that the diminution in muscle function is or might be due to the mechanical presence of the instrument rather than to free escape of air from the lower respiratory passages.

As a control the 3 previously cited tracheotomized patients were tested with and without occlusion of the tracheotomy tubes. These tests served to determine

TABLE 9.—*Results of Experiment with Bronchoscope and Hand Pulling*

	Amount of Pull, Lb.		
Males:	Normal Conditions	Bronchoscope in Situ and Open	Bronchoscope in Situ and Closed
11	98* (44.5 Kg.)	70 (32 Kg.)	82 (37 Kg.)
4	94 (42.5 Kg.)	88 (40 Kg.)	89 (40.5 Kg.)
6	98* (44.5 Kg.)	89 (40.5 Kg.)	89 (40.5 Kg.)
1	98* (44.5 Kg.)	98* (44.5 Kg.)	98* (44.5 Kg.)
5	95 (43 Kg.)	98* (44.5 Kg.)	
Females:			
3	60 (27 Kg.)	64 (29 Kg.)	60 (27 Kg.)
7	46 (21 Kg.)	52 (23.5 Kg.)	
12	56 (25.5 Kg.)	40 (18 Kg.)	44 (20 Kg.)

* Ninety-eight pounds represented the maximum registered by the scales, and it is possible that the pull exerted was above this.

TABLE 10.—*Results of Experiment with Tracheotomy Tube and Hand Pulling*

	Maximum Amount of Pull, Lb.	
Patient	Tracheotomy Tube Open	Tracheotomy Tube Closed
8	90 (41 Kg.)	90 (41 Kg.)
9	50 (22.5 Kg.)	52 (23.5 Kg.)
10	62 (28 Kg.)	60 (27 Kg.)
13	20 (9 Kg.)	14 (6.5 Kg.)

what effect on pectoral girdle action was produced by allowing free escape of pulmonary air as against the trapping of the air. However, the latter experiments, unlike those with the bronchoscope, presented the advantage of having been made without the complicating element of additional unknown factors (i. e., the effect of the presence of the bronchoscope). It will be recalled that patient 10 had a normal larynx in addition to the tracheotomy tube. The other 2 patients had completely occluded larynxes. A fourth tracheotomized subject (a boy of 6 years, patient 13) was also studied. The findings in this group are outlined in table 10.

In the tracheotomized group, likewise, there was no consistent evidence of increased pectoral girdle strength in the presence of an occluded airway as compared with a nonoccluded airway, so that closure of the trachea, either physiologic or mechanical, by a valve which traps air would seem to have no effect. This corroborates the findings in the experiment with the bronchoscope.

In the belief that possibly the position of the diaphragm might have some bearing on these results I have in 4 subjects (2 trachectomized and 2 non-tracheotomized) tested the strength of pull on inspiratory effort with the diaphragm descended and again with the diaphragm elevated in a stage of exaggerated expiration. The results are outlined in table 11, the total pull of the two arms being cited.

From a study of table 11 it can be seen that except for minor variations well within the limits of experimental error the pectoral muscle girdle has disclosed no change in strength corresponding to change in the position of the diaphragm, in the state of expansion of the thoracic cage or in the state of the airway as to occlusion or nonocclusion, mechanical or physiologic.

Conclusions Based on Observations of the Pectoral Muscle Girdle in Experiments.—The findings are rather startling, being contrary to all previous writings by a number of authorities as quoted. Certainly the majority of opinions on this subject have been based on conjecture or observations of phenomena indirectly related to the problem, and not on direct experimental evidence. There are at the same time weaknesses in my own report. The number of observations is limited. Some of the subjects are women unaccustomed to forceful pectoral girdle activity,

TABLE 11.—*Results of Experiment with Changes in the Position of the Diaphragm and Hand Pulling*

	Maximum Pull, Lb.					
	Normal Conditions		Tracheotomy Tube or Bronchoscope Patent		Tracheotomy Tube or Bronchoscope Occluded	
Subject	Inspiration	Expiration	Inspiration	Expiration	Inspiration	Expiration
7.....	52 (23.5 Kg.)	52 (23.5 Kg.)	53 (24 Kg.)	54 (24.5 Kg.)	52 (23.5 Kg.)	54 (24.5 Kg.)
3.....	72 (32.5 Kg.)	72 (32.5 Kg.)	57 (26 Kg.)	60 (27 Kg.)	60 (27 Kg.)	60 (27 Kg.)
8.....	Tracheotomized		90 (41 Kg.)	85 (38.5 Kg.)	90 (41 Kg.)	90 (41 Kg.)
9.....	Tracheotomized		77 (35 Kg.)	77 (35 Kg.)	77 (35 Kg.)	81 (36.5 Kg.)

so that possibly in them the usual physiologic reactions do not take place in the use of these muscles. Possibly the methods of testing may be considered by some as not actually a fair representation of the facts they are supposed to furnish and the resulting conclusions therefore unjustified. The scales used each registered a maximum pull of only 50 pounds (22.5 Kg.), and since some of the subjects reached the maximum or very near it, inaccuracies due to this may have colored the picture. But many of the subjects did not pull the maximum, and the record of pull in pounds in their cases at least was accurate.

In attempting to judge the situation impartially, however, I am unable to find in my experiments even a single instance which contradicts the general trend and demonstrates a direct relation between the sphincteric valvular action of the larynx and the quantitative muscular pull of the pectoral girdle. There is no doubt in my mind, therefore, that the relation between the two is not as clearcut as previously believed.

One can, however, be sure that, especially in males, physiologic closure of the sphincter valve of the larynx does take place during forceful pectoral girdle muscle contractions. This phenomenon has been photographed repeatedly by me,[15] and observed by others, cited in the beginning of this thesis. The purpose of this closure and the ultimate advantages it presents in pectoral girdle action have not yet been finally determined.

IX

SUMMARY

Part I.—The literature quoted indicates that the larynx is capable of undergoing a closure which is sphincteric in nature and which represents a means of physiologically isolating the upper from the lower respiratory passages. This closure consists of forceful approximation of the true and the false vocal cords of one side to those of the other. The sphincteric action comes into play in all expulsive acts, such as sneezing, coughing, vomiting or defecation, and in those muscular actions in which it is necessary to have the thorax fixed. It also closes during swallowing.

In addition to the closure of the true and the false vocal cords, a tier of closure is found at the level of the aryepiglottic folds.

Part II.—The literature indicates that the closure of the laryngeal sphincter is valvular in nature, the resistance it offers to pressure from above or below being far greater than would be possible from a simple contraction of the muscles involved. The false vocal cords act as one way valves preventing the egress of air from the trachea, and the true vocal cords, as one way valves preventing ingress of air.

Pressure from below serves only to wedge the false cords together more tightly, thus increasing the efficiency of the valve, and pressure from above acts in the same way on the true vocal cords.

Part III.—Original experiments demonstrated the effect of the action of the laryngeal sphincter on the movements and the position of the diaphragm. Observations on the diaphragmatic movements occurring during comparable efforts to increase intra-abdominal pressure when the laryngeal sphincter was functioning and when it was deprived of its function revealed that on effort to increase intra-abdominal pressure the concurrent upward movement of the diaphragm was on the average twice as great when the sphincteric closure of the larynx did not take place. Thus, with the airway patent the volume of the abdominal cavity is comparatively increased by virtue of the elevation of the diaphragm during efforts to increase intra-abdominal pressure. This increase in volume tends to lessen the elevation in intra-abdominal pressure, all other factors being equal.

Part IV.—Correlation of these observations and the studies of the literature demonstrates the effect of the sphincter action of the larynx on the ability to increase intra-abdominal and intrathoracic pressure and reveals the direct relation of this effect to the varied positions of the diaphragm when the airway is patent and when it is closed.

Stabilization and fixation of the diaphragm during attempts to increase intra-abdominal pressure are dependent in part on an occlusion of the airway such as occurs when the laryngeal sphincter closes. The same stabilizing effect is produced by occluding the airway at a higher level as in the Valsalva experiment, which consists of efforts to increase intrathoracic and intra-abdominal pressure by closure of the mouth and the nares. Under these conditions of respiratory occlusion a marked increase in intra-abdominal and intrathoracic pressure can be brought about, but ability to increase the pressure is lessened under circumstances in which the airway is patent and diaphragmatic excursion therefore comparatively not limited.

The anatomic structures within the larynx chiefly responsible for producing these effects are the false vocal cords.

Thus closure of the larynx plays a most important role in all expulsive acts, such as vomiting and coughing.

Part V.—Original experiments, controlled in various ways, demonstrated that the ability of a human subject to increase intra-abdominal pressure (as measured by bladder pressure) is about 19 per cent greater with than without the action of the laryngeal sphincter.

Part *VI.*—The increase of about 19 per cent in the ability to increase intra-abdominal pressure when the sphincter is closed is due essentially to fixation of the diaphragm, which in turn is dependent on trapping of air without the thorax. By this means a firm wall is established as the superior boundary of the abdominal cavity so that contraction of the abdominal parietes results in maximum increases of pressure within that cavity instead of the partial increase in volume that would occur in the presence of a mobile diaphragm. The trapping of air within the thorax by closure of the laryngeal sphincter valve prevents upward displacement of the diaphragm.

Parts VII and VIII.—The pectoral muscle girdle is said to be dependent for strength of action in part at least on fixation of the thoracic cage from which the muscles have their origin. The laryngeal sphincter valve is especially well developed in species which make extensive use of the forelimbs. Experiments showed no loss of strength in the pectoral muscle girdle when the human subject was deprived of the use of the laryngeal sphincter valve, and the theories which previous investigators had advanced relating to the action of this muscle group were not corroborated. The position of the diaphragm seemed to have no effect on these observations. However, the experiments leave room for doubt and the situation requires further study.

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NOTE.—The opinions or assertions contained herein are the private ones of the writer and are not to be construed as official or as reflecting the views of the Navy Department or the Naval Service at large.

BENIGN NONTUBERCULOUS BRONCHIAL STENOSIS

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Benign nontuberculous bronchial stenosis is a rather common complication of infections of the respiratory tract but it is rarely recognized. It is important that more study be given to this type of lesion so that diagnostic accuracy may be increased and proper treatment instituted.

HISTORY

Knowledge of obstructing bronchial lesions has developed but recently. During the eighteenth century, not much attention was given to the clinical study of bronchial obstruction other than that due to foreign bodies.[1] Pulmonary emphysema as a result of valvular bronchial obstruction was observed by Laennec,[2] who believed it was due to the imprisonment of inspired air behind an obstruction. He assumed a relative weakness in the muscles of expiration as the explanation of this phenomenon. Ingals,[2] in 1905, and Gottstein,[2] in 1907, called attention to the fact that during inspiration the bronchial lumen is increased in size and that during expiration it is narrowed. The recognition of atelectasis as a result of bronchostenosis was difficult since this finding was readily confused with pneumonia. Legendre and Bailly [2] described the acquired form of atelectasis in 1844. They noticed that bronchial obstruction was one of the circumstances favoring its formation. The full importance of this was noted in 1845, when Mendelsohn produced it experimentally in animals.[2] Fuchs,[2] in 1849, suggested that the air of an atelectatic lung was absorbed into the circulating blood. This theory was substantiated by experimental work performed by Lichtheim [2] in 1878.

The discovery of roentgen rays in 1895 and the use of direct bronchoscopy by Killian in 1898 have proved to be the two most important developments for clarifying clinical knowledge of obstructing bronchial lesions. An apparent increase in the incidence of these lesions developed as physicians became skilled in the diagnostic use of roentgen rays and bronchoscopy.

ETIOLOGY

Bronchial obstruction may be congenital or acquired.[3] The causes of bronchial obstruction have been classified according to their site.[4] Such a classification follows:

I. Extrabronchial abnormalities causing pressure on bronchi
  1. Aortic aneurysm
  2. Enlargement of tracheobronchial lymph nodes due to:
    (*a*) Inflammation
    (*b*) Metastasis of cancer
    (*c*) Lymphoblastoma

From the Division of Medicine, Mayo Clinic.

1. Lord, F. T.: Certain Aspects of the Diagnosis of Bronchostenosis, J. Thoracic Surg. 1:573-580 (Aug.) 1932.
2. Cited by Lord.[1]
3. Eloesser, L.: Bronchial Stenosis: I., J. Thoracic Surg. 1:194-213 (Dec.) 1931.
4. Lord, F. T.: Diseases of the Bronchi, Lungs and Pleura, ed. 2, Philadelphia, Lea & Febiger, 1925.

3. Cancer of bronchus or esophagus
4. Cyst
5. Pericardial effusion
6. Dilatation of left auricle due to:
   (*a*) Mitral stenosis
   (*b*) Mitral insufficiency
   (*c*) Aortic insufficiency
7. Mediastinal abscess
8. Echinococcus disease

II. Diseases of the bronchial walls

1. Neoplasm
2. Tuberculous bronchitis
3. Nonsyphilitic and nontuberculous inflammation of bronchi
4. Syphilis

III. Intrabronchial conditions

1. Fibrinous casts
2. Foreign bodies

PATHOLOGY

It is beyond the scope of this paper to describe the many lesions that can cause bronchial obstruction. As can be seen, the pathologic changes will have a wide variation. However, many of the lesions that cause bronchial stenosis have certain similar features.

Sudden total occlusion of any bronchus will cause atelectasis of the part of the lung supplied by the bronchus, owing to the absorption of air. Partial occlusion is a far more frequent lesion. With this type of lesion there may be partial atelectasis owing to diminution of the air in the part of the lung involved, or, as in the case of obstructive emphysema, one may see a ball valve type of obstruction during the expiratory phase of respiration. In cases of bronchial obstruction the bronchi distal to the site of the lesion gradually dilate. Inflammatory processes soften the walls of the bronchi and cause loss of elasticity. If infection is present distal to the point of obstruction, purulent secretion accumulates and bronchiectasis occurs. In complete aseptic obstruction the lung becomes atelectatic and there is no secretion. In most cases the picture is complicated with septic processes.

In the past, syphilitic bronchial stricture has been described. At present, much is being written about tuberculous bronchial stricture. Not much attention has been called to stricture produced by nonsyphilitic and nontuberculous inflammation of the bronchi. This type of stricture may develop secondary to a chronic bronchial infection such as that seen in cases of chronic asthmatic bronchitis. Secondary and tertiary bronchi are principally involved. Primary bronchi are occasionally involved. The pathologic changes of this lesion have not been described fully, but one must assume that the inflammatory reaction throughout the bronchial wall must have been severe enough to cause fibrosis with subsequent stricture. Stagnation of bronchial secretions probably also plays an important part in the genesis of this type of lesion.

Holinger [5] has shown that bronchial inflammation can cause bronchostenosis and finally bronchiectasis. Anspach [6] made a follow-up study of a group of cases

5. Holinger, P. H.: The Role of Inflammatory Bronchial Stenosis in the Etiology of Bronchiectasis, Ann. Otol., Rhin. & Laryng. **47**:1070-1082 (Dec.) 1938.

6. Anspach, cited by Holinger.[5]

in which roentgenologic examination disclosed evidence of a triangular-shaped shadow in the base of one or both lungs for as long as twelve years. Physical examination revealed dulness to percussion and bronchial breathing, bronchophony or absence of breath sounds over the site of the shadow. The diagnosis usually made was pneumonia, unresolved pneumonia, mediastinal or interlobar empyema or atelectasis. In some cases these abnormal findings rapidly returned to normal and the clinical course improved in a corresponding manner. Some shadows would disappear, reappear and finally disappear again. Some shadows remained more or less unchanged. In 9 cases in which the patients came to necropsy it was found that triangular-shaped shadow seen by roentgenologic study represented an atelectatic portion of the lung. In cases in which the shadow remained present for several months or a year or more, bronchiectasis developed. The bronchoscopic findings were checked with the clinical and the roentgenologic findings in some of these cases. Bronchoscopy disclosed bronchial stenosis in the cases in which this procedure was employed.

PREVENTION

Attention has been called to the importance of keeping open the normal channels of drainage in the lung.[7] Secretion normally drains well toward the oral cavity. Several agencies facilitate drainage; these are the action of cilia, coughing and possibly peristaltic movements of the bronchi. Shallow breathing predisposes to bronchial obstruction by setting up conditions which are favorable for the stagnation of secretions. Atelectasis soon develops in that portion of the lung distal to the site of obstruction. It has been maintained that deep breathing distends the bronchial tree and tends to inflate atelectatic portions of the lung. A concentration of 5 per cent carbon dioxide in oxygen can be used as a stimulus for deep breathing in any case in which there is potential danger of atelectasis.

SYMPTOMS

The syndrome of benign nontuberculous bronchial stenosis is a rather definite one. There is usually a history of antecedent pneumonia, severe tracheobronchitis or repeated seizures of asthmatic bronchitis. After variable periods, recurrent bouts of chills, fever and general malaise develop. The period of fever is usually preceded by an irritating, nonproductive cough. The temperature may reach 102 to 103 F., and the elevation may persist for two to seven days, at the end of which time variable amounts of purulent secretion are coughed up. This relieves the symptoms. At times the sputum is blood tinged. There may be pleuritic pain on the side involved. In an attack there is stagnation of infected material distal to the stricture of the bronchus. A local pneumonic process develops. Symptoms persist until drainage in the involved region is reestablished. There may be symptom-free periods of from three months to two or three years. In most cases the disease remains undiagnosed for years. Recurrent pneumonia, "flu," malaria and brucellosis are some of the diagnoses most commonly considered. Urinary infections, biliary calculi in the common bile duct, typhoid and tularemia are other diagnoses that at times are considered.

PHYSICAL FINDINGS

When a large bronchus is obstructed, either atelectasis or emphysema (ball valve obstruction) occurs in that portion distal to the obstruction. When

7. Henderson, Y.: The Physiology of Atelectasis, J. A. M. A. **93**:96-98 (July 13) 1929.

emphysema is confined to one lung, there is an increase in the size of the side affected, with widening of the intercostal spaces. On percussion the note is hyperresonant or tympanic. Auscultation reveals breath sounds diminished or absent. The whispered voice and tactile fremitus are diminished or absent. The heart and the mediastinum are displaced away from the affected side, and the diaphragm on the affected side may be displaced downward.

In cases of obstructive atelectasis there may be a respiratory lag and diminished excursion of the thorax on the affected side. There may be dulness to percussion, and the breath sounds may be decreased in intensity or may be absent. The heart and the mediastinum are drawn to the affected side, and the diaphragm is elevated on this side. Partial bronchial obstruction may yield few or no definite physical findings.

Eloesser [8] said that there may be distinct retraction of the thoracic wall over the atelectatic portion of the lung. He said that this is especially true in cases in which the stenosis involves a bronchus supplying a lower lobe. "Not only the intercostal spaces but the whole chest wall, over an area the size of a hand, may be retracted on inspiration while the rest of the chest expands normally." He said that this sign is pathognomonic of bronchial stenosis.

Benign nontuberculous bronchial stenosis is more likely to occur in the lower lobe than in the upper lobe or in the middle lobe of the right lung. Physical findings may be absent, since the stenosis usually involves the third division of a bronchus. If physical findings are present, they usually are those of obstructive atelectasis, namely, dulness on percussion and decrease in breath sounds over that portion of the lung supplied by the involved bronchus.

ROENTGENOLOGIC FINDINGS

In obstructive atelectasis the physical findings may be of more value than the roentgenologic findings. Roentgenologic examination is important in determining the presence of atelectasis, but the roentgenologic findings alone will not always enable one to determine whether or not a bronchus leading to the affected region is open or closed.

The roentgenologic findings in obstructive atelectasis may be listed as follows: The pulmonary field is reduced on the affected side; the heart and the mediastinum are displaced toward the affected side; there may be narrowing of the intercostal spaces with increased downward inclination of the ribs, and there are elevation and decreased motion of the diaphragm on the involved side. The atelectatic region itself will show increased density. The trachea is displaced toward the side of the lesion. On inspiration the heart is drawn toward the affected side, and on expiration the mediastinum moves away from this side.

In cases of valvular obstruction with emphysema the roentgenograms show increased transparency of the involved side. The diaphragm is depressed and partially fixed. The mediastinal structures are displaced to the opposite side from that of the lesion. There is increased movement of the diaphragm on the unaffected side. These signs are accentuated at the end of expiration. If a small bronchus is involved, the roentgenologic findings may be negative. Sometimes a small triangular shadow with its apex pointing toward the hilus is seen in one of the cardiophrenic angles.

Bronchography may help to determine whether or not an obstruction exists and to localize the site of obstruction. It also aids in demonstrating the condition of adjacent pulmonary parenchyma; it does not, however, permit determination of the

8. Eloesser, L.: Bronchial Stenosis: III., J. Thoracic Surg. **1**:373-396 (April) 1932.

cause of bronchostenosis, and it is for this reason that bronchoscopy should be performed in every case of suspected bronchostenosis if the general physical condition of the patient will permit.

BRONCHOSCOPIC FINDINGS

Bronchoscopy is the only clinical method of determining whether or not benign bronchial stenosis exists. It is the only way by which such a lesion can be satisfactorily treated. It is wise to wait two or three weeks after recovery, if there has been a recent acute phase of the disease, before employing bronchoscopy in any case in which such a lesion is suspected. If this procedure is followed, one may see a normal tracheobronchial tree except for the involved bronchus. If the patient is suffering from chronic asthmatic bronchitis, the bronchial mucosa is inflamed and variable amounts of secretion are present on both sides. This secretion may be mucoid, mucofibrinous or mucopurulent. The stenotic bronchus is almost always a branch of one of the bronchi supplying the lower lobe. The mucous

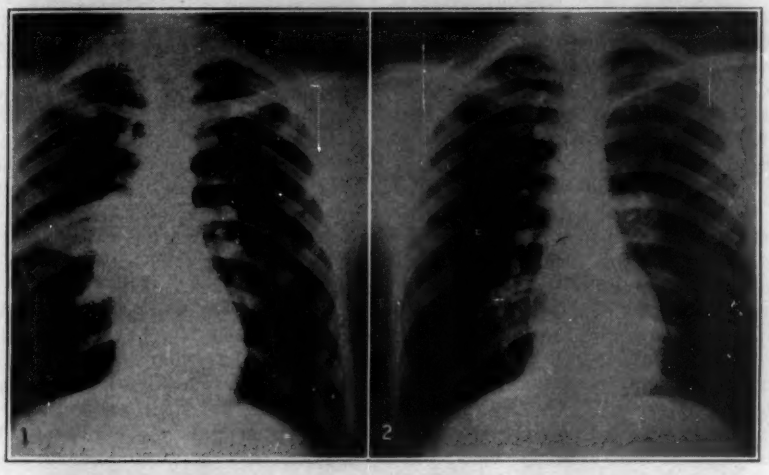

Fig. 1 (case 1).—Roentgenogram of lungs before bronchoscopic dilation.
Fig. 2 (case 1).—Roentgenogram of lungs after bronchoscopic dilation.

membrane around the orifice is inflamed and edematous. The normal diameter of the orifice may be reduced to a sixth or a fourth, or the bronchus may be completely closed. It has a rigid appearance; that is, it does not increase in size during inspiration and decrease in size during expiration. Almost always there is a trickle of purulent secretion coming from its orifice. If the bronchus is dilated, bloody mucopurulent material comes from the depths of the bronchus. The bronchus bleeds more readily when touched than does a normal bronchus. At times it is difficult to be absolutely certain that the lesion is not a cancer, and it is advisable to obtain a specimen of tissue for microscopic diagnosis.

This paper is based on a study of 25 cases of benign nontuberculous bronchial stenosis in which bronchoscopy was performed by me. I shall report 4 of these cases, which appear to be of especial interest, and shall review the findings in the entire series of cases.

REPORT OF CASES

Case 1.—A boy 15 years of age had suffered from recurrent attacks of "bronchial colds" since childhood. In the spring of 1937 he had begun to have recurrent attacks of chills, fever, general malaise and a nonproductvie cough. During these attacks his temperature had risen to

102 F. and had remained at that level four or five days. He had been forced to stop school because of these attacks. Typhoid had been suspected, but agglutination tests had all been negative. He had lost 13 pounds (5.9 Kg.) in one year.

Physical examination of the thorax did not reveal any abnormality. Roentgenographic study of the lungs revealed a triangular-shaped infiltration extending from the right hilus (fig. 1). Roentgenographic examination of the paranasal sinuses revealed thickening of the lining membrane and marginal sclerosis of the left maxillary sinus. The sinuses were investigated, with negative results. Repeated examination of the sputum failed to disclose Mycobacterium tuberculosis.

Bronchoscopy revealed evidence of bilateral bronchitis. A bronchus arising posteriorly and laterally from the right stem bronchus was found to be stenotic. This was dilated and a moderate amount of bleeding occurred. Bronchography subsequently revealed some dilatation of the bronchus. The patient made a satisfactory recovery and gained 13 pounds (5.9 Kg.) in three months. The roentgenographic appearance of the lung became normal (fig. 2). In a letter written four years after the completion of bronchoscopic treatment, the patient said that he was well.

CASE 2.—The patient was a man aged 47 years. Three weeks before he came to the clinic he had had a cough which had produced large amounts of purulent material. One week later a pleuritic type of pain had developed over the left side of the thorax. The day before he registered at the clinic he had had a chill and fever. On the day of registration his temperature was 99 F. During the next two weeks it ranged from normal to 101.4 F. He would raise from 30 to 310 cc. of foul-smelling purulent sputum in twenty-four hours.

Physical examination revealed impaired resonance and some decrease in the intensity of the breath sounds over the lower lobe of the left lung. Roentgenograms of the thorax showed an area of density over this lobe which extended superiorly as far as the fourth rib. The left half of the diaphragm was elevated (fig. 3). Repeated examination of the sputum showed no evidence of pneumococci or of M. tuberculosis. The clinical picture was that of acute infection and atelectasis involving the lower lobe of the left lung. Postural drainage exercises were started. Bronchoscopy, which was performed six days after the patient was admitted to the hospital, disclosed purulent secretion coming from all the divisions of the bronchus supplying the lower lobe of the left lung. The orifices of these smaller bronchi had been reduced in size by an inflammatory edema of the mucous membrane. A lateral division of the bronchus supplying the lower lobe of the left lung was almost closed. Several divisions of the bronchus supplying the lower lobe of the left lung were dilated by means of dilating forceps and thoroughly aspirated. Bronchoscopic dilation was repeated on two subsequent occasions.

The patient improved slowly. He was dismissed from the hospital twenty days after his admission. At this time he was afebrile but was raising large amounts of purulent secretion. Bronchography later revealed bronchiectasis of the lower lobe of the left lung (figs. 4 and 5). Clinical improvement continued. Five months after the onset of his illess, he had gained 30 pounds (13.6 Kg.) and was almost free of symptoms referable to the lung. At that time he was raising only small amounts of mucoid secretion. He was free of symptoms when he returned to the clinic for reexamination nine months later. At this time bronchography did not disclose any abnormality on the left side (fig. 6). The patient had made a complete recovery from an extremely serious and disabling illness.

CASE 3.—A boy aged 11 years, the son of a physician, came to the clinic Oct. 5, 1938. He had had asthma since 1930. The attacks of asthma usually had been associated with infection of the respiratory tract. In September 1934 and in the fall of 1935 he had had pneumonia of the lower lobe of the right lung. In the fall of 1936 he had had bilateral bronchopneumonia. Since that time he had not been well. He had had frequent colds, which had been associated with asthma. He had felt tired and had had a persistent afternoon rise of temperature, which had ranged from 99 to 99.6 F. Three weeks before he came to the clinic his temperature had reached 102 F.

Physical examination revealed dulness and bronchial breathing over the inferior portion of the lower lobe of the right lung. Roentgenographic examination disclosed infiltration and partial atelectasis of this lobe (fig. 7). Roentgenoscopy disclosed limitation of movement of the right half of the diaphragm. Examination of the sputum did not reveal M. tuberculosis. Otolaryngologic examination did not disclose any abnormality. Roentgenograms of the paranasal sinuses were normal.

Bronchoscopy disclosed stenosis of a posterior division of the bronchus supplying the lower lobe of the right lung. A rather large amount of purulent material was coming from the orifice of this bronchus. The stenotic bronchus was dilated. The patient was told how to

employ postural drainage exercises and was advised to spend the following winter in Arizona. He was seen nine months after he had left the clinic. He was free of symptoms and had gained 8 pounds (3.6 Kg.). Roentgenographic examination of the thorax did not disclose any abnormality at this time. In a letter written four years later, the patient's father said that bronchoscopy had been performed several times at home and that the patient had had occasional attacks of wheezing and cough associated with some fever. He stated in his letter: "We are well pleased with his condition and feel that he was markedly benefited by the bronchoscopies." The family has moved to Arizona.

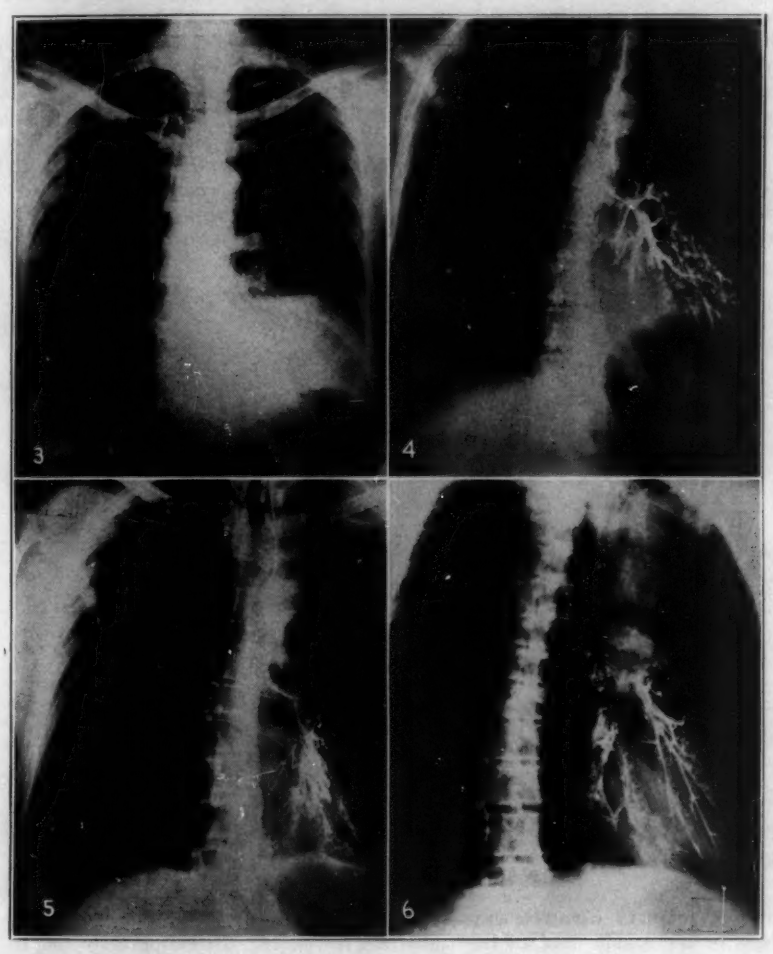

Fig. 3 (case 2).—Roentgenogram of the lung on admission to the hospital, showing the left side of the diaphragm elevated and the lower lobe of the left lung infiltrated throughout.

Fig. 4 (case 2).—Bronchogram of the lower lobe of the left lung made two weeks after admission, showing marked dilatation of the left lower lobe bronchus and its branches.

Fig. 5 (case 2).—Bronchogram made five months later, showing slight residual dilatation of some of the branches of the bronchus supplying the lower lobe of the left lung. The left half of the diaphragm approximates its normal position.

Fig. 6 (case 2).—Bronchogram showing normal diameter of the branches of the bronchus supplying the lower lobe of the left lung.

CASE 4.—A girl aged 14 years came to the clinic June 23, 1942. A dry, nonproductive cough had developed in April 1942. This had lasted for about a week and then had disappeared almost completely. Since that time she occasionally had a nonproductive cough. On April 28, 1942 roentgenographic examination had disclosed a triangular region of density

in the right lung. Secondary atelectasis resulting from the presence of a nonopaque foreign body had been suspected. On June 11, 1942 the roentgenologic findings had been the same as they had been previously. During this period of observation the patient had taken her temperature three times each day but she never had had any fever.

Physical examination at the clinic revealed that the intensity of the breath sounds was decreased over the middle lobe of the right lung. Roentgenographic examination disclosed a triangular region of increased density over the anterior and inferior portions of the right lung (fig. 8). The roentgenologist expressed the opinion that this was due to atelectasis of the middle lobe of the right lung. Examination of the sputum failed to disclose M. tuberculosis.

Bronchoscopy revealed that the bronchus supplying the middle lobe of the right lung had been closed completely by an inflammatory stricture. The stenotic bronchus was dilated, and aspiration produced 50 cc. of mucopus. Four days later roentgenographic examination did not reveal any change in the appearance of the right lung. Bronchoscopy disclosed that the bronchus supplying the middle lobe of the right lung was almost completely closed. A moderate amount of pus was coming from the orifice of the bronchus. The stenotic bronchus again was dilated, and a moderate amount of mucopus was aspirated. This was used to inoculate guinea pigs, but it evidently did not contain M. tuberculosis. A partial pneumothorax developed, and the right lung became about half its normal size. This caused the

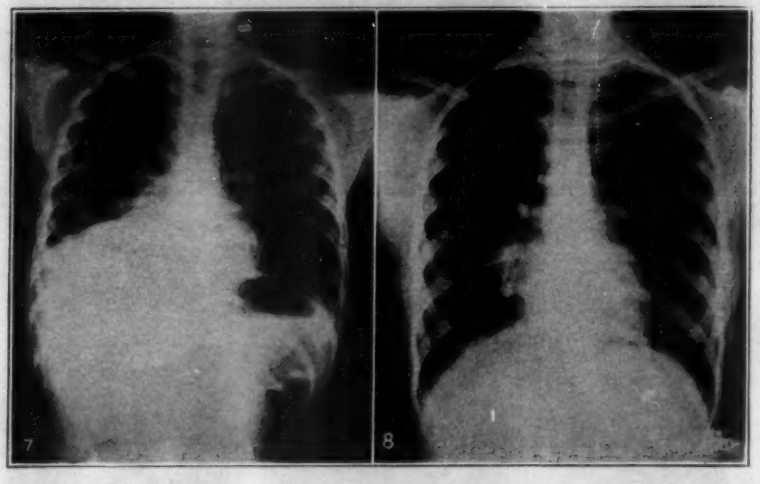

Fig 7 (case 3).—Roentgenogram of the thorax showing evidence of partial atelectasis of the lower lobe of the right lung.

Fig. 8 (case 4).—Roentgenogram of the thorax showing atelectasis of the right middle lobe of the right lung.

patient little discomfort. Complete expansion of the lung occurred in one month, but evidence of atelectasis of the middle lobe of the right lung persisted. Bronchography did not disclose any evidence of bronchiectasis. A recent roentgenogram shows the middle lobe of the right lung to be reexpanded, and the local physician has written stating that this patient has done well.

COMMENT

In a review of the 25 cases of benign nontuberculous bronchial stenosis seen by me there are certain features which are worthy of note. Twelve of the patients were males and 13 were females. The oldest patient was 61 years of age, and the youngest was 6 years of age. The average age of the patients was 34 years. In 15 of the 25 cases bronchial stenosis was secondary to asthma or asthmatic bronchitis. In 2 of these 15 cases the asthma apparently was allergic; in 6 cases it apparently had an infectious background. In 7 cases there was a history of infection and of an allergic disturbance. Almost all of the patients had had one or more rather

severe attacks of infection of the respiratory tract. Table 1 shows the infections that had occurred in these cases.

It seems safe to state that severe infection and chronic irritation of the tracheobronchial tree are important etiologic factors in this type of bronchial stenosis.

The symptoms in this series of cases are listed in table 2. Six patients had lost between 20 and 30 pounds (9.1 and 13.6 Kg.) and 3 had lost between 10 and 20 pounds (4.5 and 9.1 Kg.) during the course of their illness. The physical findings are listed in table 3.

The roentgenographic appearance of the lungs was reported as being normal in 11 cases, and abnormal findings were obtained in 14 cases. Most of these patients were not seen during an acute episode of retention of bronchial secretion and secondary pneumonitis. It is highly possible that if they had been examined during

TABLE 1.—*Past History of Infectious Disease*

Disease	Cases	Disease	Cases
Pneumonia	12*	Scarlet fever	5
Influenza	10	Diphtheria	4
Measles	5	Malaria	1
Chickenpox	5	Typhoid	1
Whooping cough	4	Abscess of lung	1

* Three attacks occurred in one case and two attacks in another.

TABLE 2.—*Symptoms*

Symptoms	Cases		Symptoms	Cases
Cough	23		Fever	17
Sputum			Attacks of asthma	15
Purulent	10		Chills	8
Purulent and bloody	9	21 cases	Dyspnea on exertion	5
Mucoid	2		Sweating	4

TABLE 3.—*Physical Findings*

Findings	Cases
Impaired resonance and decrease in intensity of breath sounds over portion of lung supplied by involved bronchus	11
Generalized wheezing and rales but no localized findings	7
Normal	5
A few moist rales over portion of lung supplied by involved bronchus	1
Consolidation over portion of lung supplied by involved bronchus	1

TABLE 4.—*Results of Nasolaryngologic Examination*

Results	Cases
Normal	4
Active sinusitis	4
Roentgenographic evidence of previous sinusitis but no clinical evidence of active sinusitis	4
Infected tonsils	3
Allergic mucous membrane	3
Examination not performed	4
Nasal obstruction	2
Mucous cyst on floor of maxillary sinus	1

such a period the physical and roentgenographic examinations of the lungs would have disclosed changes in all instances.

Most of the patients were seen during a so-called quiescent period. Because of this, the temperature was normal in all but 8 cases. The oral temperatures of 6 patients ranged from 99 to 102.4 F. The average leukocyte count was 10,000 per cubic millimeter of blood. In 10 cases the leukocyte counts were about 10,000. The highest count was 19,000 leukocytes per cubic millimeter of blood. Anemia was an unusual finding. The average value for the hemoglobin was 15.1 Gm. per hundred cubic centimeters of blood. The values fell below 13 Gm. in 2 cases. In these cases the values were 12.7 and 11.2 Gm., respectively. The sputum did not contain M. tuberculosis in any instance

The importance of recognizing infections of the upper part of the respiratory tract has long been recognized in regard to nontuberculous bronchial infection. The results of examinations of the nose and throat are shown in table 4.

The stenosis almost always involved a division of the bronchus supplying a lower lobe.

An analysis of the results of treatment necessarily must be based on the 19 cases in which follow-up data are available. In most of these cases bronchoscopy was performed from three to six years before this report was made. Four patients obtained complete relief of symptoms; 6 patients obtained great relief, which has persisted up to the present time; 3 patients obtained marked relief but had a recurrence of symptoms twenty-four, twenty-five and thirty-six months, respectively, after bronchoscopy; 1 patient obtained moderate relief for two and a half years and then had a recurrence of symptoms; 1 patient has had moderate relief for three years and three months, and 1 patient obtained relief for four months and then had a recurrence of symptoms. The remaining 3 patients apparently were not benefited by the treatment.

SUMMARY

Benign nontuberculous bronchial stenosis frequently remains undiagnosed for a long time. An antecedent infection of the respiratory tract, such as pneumonia, severe tracheobronchitis or asthmatic bronchitis, can cause bronchial obstruction. After stenosis has occurred it produces a rather typical clinical picture which is characterized by recurrent chills and fever, cough, purulent secretion and general malaise. The physical and roentgenographic findings vary. In some cases they indicate partial atelectasis of a lower lobe of one lung. In other cases they may be negative. Bronchoscopy is the only accurate method of diagnosing such a lesion. Bronchoscopic dilatation produces satisfactory results in about 66 per cent of cases. In a certain number of cases the stenosis recurs and it is necessary to repeat bronchoscopic dilatation.

CANCER OF THE LARYNX

A RADIOTHERAPEUTIC TEST AS AN AID IN CHOOSING BETWEEN OPERATION AND IRRADIATION

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The purpose of this communication is to present a radiotherapeutic test that has proved to be of practical value in testing the radiosensitivity of cancer of the larynx. The information thus gained is distinctly useful in deciding between laryngectomy and radiotherapy in certain cases of intrinsic, operable carcinoma in which a decision is otherwise difficult.

Therapeutically, cancers of the larynx appear to fall into five general clinical groups: (1) clearly inoperable lesions; (2) clearly operable early lesions suitable for laryngofissure or radiotherapy; (3) clearly operable lesions too advanced for laryngofissure or radiotherapy but suitable for laryngectomy; (4) lesions which are too advanced for laryngofissure but suitable for either laryngectomy or radiotherapy in regard to which a choice between the two procedures is difficult because partial mobility of the structures indicates probable radiosensitivity; (5) cancers in patients in whom certain general conditions lead one to consider radiotherapy in preference to laryngectomy, such as advanced age, poor life expectancy, concomitant disease or a psychologic state adverse to laryngectomy.

This presentation limits itself to a consideration of groups 4 and 5: operable lesions in which a choice between laryngectomy and irradiation is difficult because of such local or general conditions as those indicated. A discussion of problems involved in the other groups will be the subject of a separate communication.

In groups 4 and 5 two important questions arise: 1. Is it feasible to administer a complete course of radiotherapy and resort to laryngectomy if the disease persists or recurs? 2. Is it feasible to test the radiosensitivity of a lesion by a partial course of radiotherapy as a practical guide in deciding between surgical removal and irradiation in doubtful cases?

IS IT FEASIBLE TO ADMINISTER A COMPLETE COURSE OF RADIOTHERAPY AND RESORT TO LARYNGECTOMY IF THE DISEASE PERSISTS OR RECURS?

The difficulties associated with surgical procedures performed on previously irradiated tissues are well known. Irradiation of the blood vessels and connective tissues injures them and thus impairs the circulation and delays normal healing. These changes may also render the operative area more susceptible to infection. The degree and the duration of this damage of tissues depend on the extent and the type of radiation employed. In the early days of radiotherapy severe damage to normal tissues occurred frequently. Telangiectasis, atrophy and induration of the skin and subcutaneous tissues were common. An important feature of the improvement in radiotherapy has been the better preservation of the tissues constituting the tumor bed. Thus, the ideal form of radiotherapy is specific in the sense that it destroys the neoplasm without causing permanent damage to the

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connective tissue and blood vessels of the tumor bed. This progress has been accomplished by increasing the voltage of roentgen rays, by increasing the filtration, by using low roentgen intensity, by using the smallest possible cutaneous portals and by using the penetrating gamma ray of radium when this method of treatment is indicated.

The proper relationship between daily dose, total dose and size of portals has been developed and improved with the view of producing the maximum injury to the tumor and the minimum damage to the normal tissues. The results of these advances in radiotherapy have been a marked increase in the number of cures of certain forms of cancer and a definite diminution in radiation injuries to the normal tissues. Surgeons who have had experience with operations on tissues irradiated by the older methods of radiotherapy should take these advances into consideration.

Details of Four Cases in Which Carcinoma of the Larynx Was Treated by Laryngectomy Following a Full Course of Roentgen Therapy

Case	Lesion	Type of Radiation *	Interval Between Last Irradiation of Lesion and Operation	Postoperative Course
1	Squamous carcinoma of right true and false cords, with subglottic extension and partial fixation of right hemilarynx	5,400 r in 11 days by 1 portal	46 days	Small pharyngeal fistula, healed 30 days after operation
2	Squamous and transitional cell carcinoma of right true and false cords and right aryepiglottic fold, with fixed hemilarynx	4,975 r in 11 days	60 days	Normal
3	Squamous carcinoma of right true and false cords with subglottic extension and complete fixation of right hemilarynx	5,500 r in 11 days	75 days	Slight superficial infection of wound, healed after 20 days
4	Squamous carcinoma of left true and false cords with subglottic extension and almost complete fixation of left hemilarynx	5,400 r in 11 days	117 days	Superficial infection of wound, healed after 25 days

* The other factors were: voltage, 400 kilovolts; distance, 84 cm. to 115 cm.; portals, 25 to 9 sq. cm.; filtration, 5 mm. of copper; roentgen intensities, 4.2 r to 6.3 r per minute. All measurements were made on the skin. Two treatments were given daily. The total dose was given through one portal.

In order to gain direct information on this problem in relation to the larynx, my colleagues and I have studied a group of cancers of the larynx treated by the latest methods of roentgen therapy followed by laryngectomy. The lesions had been diagnosed as carcinoma and were selected with great care as being too extensive for laryngofissure but apparently not beyond cure by laryngectomy. Roentgen therapy was used as the initial procedure for one or more of the following reasons: 1. There was partial mobility of the structures leading to the possibility of adequate radiosensitivity for cure by irradiation. 2. The lesion was a small cell plexiform epidermoid carcinoma of the transitional cell type. 3. The patient was of an advanced age or in a poor general condition. 4. The patient requested or insisted that radiotherapy be tried as the initial procedure.

The accompanying table gives data on 4 patients on whom laryngectomy was performed following complete radiotherapy.

The members of the surgical staff who performed the laryngectomies hold the following opinion [1]: No special technical difficulties were found at operation that

1. Most of the laryngectomies were done by Dr. L. B. Bernheimer.

could be attributed to irradiation of the region. There was perhaps somewhat more delay in healing of the operative wound in the irradiated area, but it is of no great consequence as all of the wounds finally healed satisfactorily. It must be recalled that the operative wounds in the absence of irradiation rarely heal by first intention. In that connection, attention is called to Brunschwig's [2] experience with panlaryngectomy following irradiation which is in general accord with our observations. He found no operative or postoperative complications due to previous irradiation.

IS IT FEASIBLE TO TEST THE RADIOSENSITIVITY OF A CANCER OF THE LARYNX BY A PARTIAL COURSE OF RADIOTHERAPY AS A PRACTICAL GUIDE IN DECIDING BETWEEN LARYNGECTOMY AND IRRADIATION IN DOUBTFUL CASES?

The idea of using a limited course of radiotherapy to test the radiosensitivity of a tumor is not new; thus a partial course of radiotherapy may be given to a mediastinal mass in order to test its radiosensitivity. Lymphosarcoma generally undergoes rapid and marked regression, whereas bronchial carcinoma usually shows little or no response to a comparatively small dose of radiation. The test is obviously more useful in differentiating between lesions in which the difference in radiosensitivity is marked.

Carcinomas of the larynx exhibit a wide range of radiosensitivity; hence they should lend themselves to a therapeutic test. The principles of such a test are that the irradiation should be adequate, should be administered over a comparatively short period, should be of a type that can be supplemented to complete a cycle that is known to be effective, and should be such that it can be followed by laryngectomy if the lesion proves to be more suitable for operation. The question naturally arises whether it is not more practical to administer the full cycle of radiotherapy rather than a partial cycle. I think this depends on the circumstances in the individual case and also on what further experience one will gain from the two methods.

In 1941 I [3] reported a new technic for the administration of external radiation which appeared to be more effective with the more radioresistant forms of cancer of the mouth, pharynx and larynx. It was called the "method of concentration." One group of patients was treated by the "interrupted method," which consisted of two cycles of roentgen therapy separated by an interval of eleven to fifteen days. The evidence so far indicates that the technic is fully as effective as the continuous method of irradiation.

At the end of twenty-one days from the beginning of treatment, or fifteen days from the last treatment, it has been possible to judge the response and estimate the relative radiosensitivity of the lesion with reasonable accuracy. The information thus gained has been useful in deciding whether to continue irradiation or to resort to laryngectomy. If the lesion has shown little or no response, the second cycle of radiotherapy is omitted, and laryngectomy is performed four to six weeks later. If the response has been marked, the second cycle of radiotherapy is administered. Actually, some of our best results have been obtained by the interrupted method of irradiation.

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2. Brunschwig, A.: Panlaryngectomy for Advanced Carcinoma of the Larynx, Surg., Gynec. & Obst. **76**:390-394 (April) 1943.

3. Cutler, M.: Concentration Method of Radiotherapy for Cancer of the Mouth, Pharynx, and Larynx, J. A. M. A. **117**:1607-1610 (Nov. 8) 1941.

We have treated cancer by this method in 25 cases, of which the following is a typical example:

A man aged 43 years complained of continuous and increasing hoarseness for one year and of pain on swallowing for three months. Examination disclosed that the right true and false cords were the seat of a large ulcerated mass projecting into the lumen of the larynx, with almost complete fixation of the right hemilarynx. The biopsy showed squamous carcinoma. For six days (Feb. 24 to March 1, 1941) the patient received two roentgen ray treatments with the various factors as follows: voltage, 400 kilovolts; filter, 5 mm. of copper; distance, 85 cm.; amperage, 5 milliamperes; portals, 30 to 20 sq. cm.; dosage, first day 400 r, second day 500 r, third day 550 r, fourth day 650 r, fifth day 700 r, sixth day 800 r, in two treatments daily. The total dose was 3,600 r, measured on the skin. The roentgen intensity varied between 6.3 r per minute and 8.9 r per minute. The first cycle of treatment was given over the right side of the larynx. On March 12, 1941, eleven days after completion of the first cycle, the lesion showed the first sign of regression and there was more mobility of the right hemilarynx. Two days later, the thirteenth day after the last treatment, there were marked regression and increased mobility.

Because of this pronounced improvement, it was decided to administer the second cycle of roentgen ray treatments. The interval between the two cycles was twelve days. The second cycle was begun March 14 and was finished March 17, with the following factors: voltage, 400 kilovolts; filter, 5 mm. of copper; distance, 85 cm.; amperage, 5 milliamperes; portals, 30 to 12 sq. cm.; dosage, first day 850 r, second day 950 r, third day 1,050 r, fourth day, 1,150 r, in two treatments daily. The total dose was 4,000 r, measured on the skin. The total dose of the second cycle was given over the left side of the larynx.

The total dose of the first cycle (3,600 r) and that of the second cycle (4,000 r) made a final total of 7,600 r for the ten treatment days.

The maximum reaction on the skin consisted of a deep pigmentation. The maximum epithelial reaction was a very mild *epithélite*.[3a] The mobility of the right hemilarynx was restored to normal on April 4, eighteen days after the completion of the second cycle of roentgen therapy. The lesion continued to regress and finally disappeared on April 25, thirty-eight days after the last treatment of the second cycle, but there remained a small irregular nonulcerated nodule about 6 mm. in diameter on the posterior extremity of the right true cord. On May 9, fifty-three days after the last roentgen treatment, it was decided to administer a third course of roentgen therapy. It was not possible to decide clinically whether or not this nodule contained a remnant of carcinoma, and it was decided that a biopsy should not be done. From previous experience it was our belief that a third course of roentgen treatment could be given with safety. Consequently, the third course was administered in two days with the various factors as follows: field, the right side of the larynx; voltage, 400 kilovolts; filters, 5 mm. of copper; distance, 80 cm.; portal, 7 sq. cm.; intensity, 7.4 to 9.8 r per minute; dosage, 500 r twice daily for two days, or a total of 2,000 r, measured on the skin. The grand total for the three cycles of roentgen therapy was 9,600 r. The last treatment was given on May 10, 1941. The nodule regressed slowly and finally became a small fibrotic thickening about 3 mm. in diameter, which has remained stationary for two years. It is now obvious that this is an area of scar tissue. On April 29, 1942 the patient had gained about 20 pounds (9 Kg.), his voice was normal, and the right hemilarynx was normal in appearance and mobility.

There are several significant features to this case. The most important is the disappearance of an extensive intrinsic squamous carcinoma of the larynx with almost complete fixation of the hemilarynx, with freedom from recurrence for over two years. Several features of the treatment are interesting. The large total dose (9,600 r) was administered in only twelve treatment days with little reaction in the skin or the mucous membrane. The division of the treatment into three cycles with two intervals afforded an opportunity to observe the response of the lesion and plan further treatment accordingly. Finally, this case illustrates that it is practical to test the radiosensitivity of a lesion with a partial course of roentgen therapy and use such information as a guide to further treatment.

3a. An *epithélite* is a reaction in the mucous membrane produced by irradiation. It consists in a destruction of the epithelium and replacement of this tissue by a grayish fibrinous membrane. The connective tissue and the blood vessels remain relatively intact. The word was coined by the Curie Institute group.

COMMENT

Until 1922 the results of radiotherapy in laryngeal cancer were so unsuccessful that surgical removal was the only form of treatment for operable lesions. In 1922 Regaud, Coutard and Hautant [4] presented 6 cases of inoperable cancer treated successfully by roentgen therapy and this was followed by other favorable reports from the Curie Institute of Paris. The trend of the problem is indicated clearly in a paper in 1938 by Jackson and Jackson [5] in which they said,

> . . . Since the year 1930 . . . our observations have led us to believe that there are growths classed as operable for which the patient is justified in choosing irradiation in preference to operation.
>
> . . . As between laryngectomy and irradiation we are decreasing the number of laryngectomies in proportion to the number of patients treated with irradiation. We find irradiation better for many patients for whom a very few years ago we would have advised laryngectomy.
>
> . . . Our experience is sufficient to warrant the belief that the future will probably see a progressive decrease in the relative number of laryngectomies.

Similar views have been expressed by Quick,[6] Lenz [7] and others for some years.

As long as radiotherapy was limited to inoperable cancers, the problem of choosing between operation and irradiation was relatively simple. Now that irradiation is conceded to be the method of choice for certain operable lesions, the problem has become much more difficult, and the responsibility for making a proper choice has greatly increased. With this change has come increased necessity for careful clinical and pathologic research in an effort to explore every factor that may help one in this decision. In rare instances the microscopic structure of the biopsy specimen is of some help, but the information thus gained is of limited value, and the interpretation is hazardous even when supported by much experience. At best the biopsy can be interpreted only in the light of the clinical findings.[8] With the exception of the radiotherapeutic test, the gross features of the lesion as seen on indirect and direct examination give the most reliable indications of radiosensitivity. The more movable the lesion and surrounding structures, the more likely is it to be radiosensitive and curable by radiotherapy. Infiltrating lesions that invade the underlying muscle and cause complete fixation are generally radioresistant, but here also there occur exceptions. Notable examples are those lesions of the ventricle that destroy the thyroid cartilage and invade the preepiglottic space and sometimes perforate the soft tissues of the neck. These lesions are frequently radiosensitive and sometimes cured in spite of their infiltration and extent. One patient with cervical metastasis belonging to this group lived eight years without recurrence and died of cardiac disease; another is now alive six years after treatment. Both were treated by intensive irradiation.

The responsibility of advising radiotherapy instead of laryngectomy under certain conditions in which the opposing factors appear to be equally balanced is indeed a serious matter. The knowledge that if radiotherapy fails, laryngectomy

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4. Regaud, C.; Coutard, H., and Hautant, A.: Rapport sur la Curie therapie et la roentgentherapie dans le cancer du larynx, Ann. d. mal. de l'oreille, du larynx **41**:967-969, 1922.

5. Jackson, C., and Jackson, C. L.: Cancer of the Larynx, J. A. M. A. **111**:1986-1993 (Nov. 26) 1938.

6. Quick, D.: Carcinoma of Larynx: Janeway Memorial Lecture, Am. J. Roentgenol. **38**:821-853 (Dec.) 1937.

7. Lenz, M.: Demonstration of Microscopically Proven Epitheliomas of Larynx Treated at Presbyterian Hospital of New York During 1931 and 1932, Laryngoscope **48**:278-279 (April) 1938.

8. Cutler, M.: Cancer of the Larynx: Relation Between Gross Anatomy, Microscopic Structure and Radiosensitivity, J. A. M. A. **115**:1339-1345 (Oct. 19) 1940.

may still be performed with safety is of considerable help. As one's experience accumulates, the decision becomes less difficult. Certain intrinsic lesions of the true vocal cord with subglottic extension and complete fixation have, with isolated exceptions, failed to respond to the most intensive radiotherapy. Consequently, the patient permitting, laryngectomy is performed if there are no general contraindications. On the other hand, I find more and more cases in which fixation of the cords is only partial and which have yielded excellent results with the present methods of irradiation. The details of these results are in preparation and will be published shortly.

The therapeutic test is merely a further effort to help one choose the best treatment for the particular cancer under consideration. The greatest care must be exercised not to withhold laryngectomy when that procedure appears to offer the best chance of cure. At the same time, due cognizance must be taken of the advances and the results of modern radiotherapy in order to give the patient the advantage of cure without laryngectomy if the circumstances warrant.

SUMMARY

The radiotherapeutic test presented has proved to be of value in testing the relative radiosensitivity of carcinoma of the larynx. The information gained by use of this test is of practical value in helping to decide between operation and irradiation under circumstances in which a decision is otherwise difficult. If the lesion shows a favorable response, the second cycle of treatment is administered; otherwise, laryngectomy is performed four to six weeks after the completion of the therapeutic test. The therapeutic test is the first phase of the "interrupted method" of treatment under the principle of concentration radiotherapy reported in a previous communication. Further experience with the use of this technic has been satisfactory and has confirmed the original opinion concerning its effectiveness.

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NEW CONTRIBUTIONS ON THE SO-CALLED OTOSCLEROSIS OF CHICKENS

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INTRODUCTION AND HISTORICAL REVIEW OF THE PROBLEM

The promising results of the operative treatment of otosclerosis by fenestration of the labyrinth have lately shifted the main interest of otologists from the question of the cause to the question of the therapy of otosclerosis. Nevertheless, from a scientific point of view the investigation of the causal genesis remains the central problem.

Many attempts have been made in this direction, and it appears that animal experiments are most likely to bring one nearer to a solution of this difficult problem. It is the general purpose of these experiments to produce changes similar to otosclerosis in the labyrinthine capsule in certain animals. By careful analysis of all the factors concerned in the production of these changes definite conclusions might be drawn as to the genesis of the human disease.

The main difficulty with which experimenters are confronted is the fact that in human otosclerosis the changes are apparently confined to the labyrinthine capsule, the rest of the skeleton, according to present knowledge, remaining free. For this reason all the experiments in which labyrinthine changes have been produced together with a generalized affection of the skeleton might not bring any decisive contribution to the solution of the problem. Otosclerosis is a localized disease, and all the experimentally produced changes have been manifestations of a generalized disease, no matter how similar their histologic picture was to that of otosclerosis. Disregard of this fundamental fact often has led to faulty conclusions. Wittmaack tried to produce more or less localized changes of the labyrinthine capsule. His experiments were based on his theory as to the cause and histogenesis of human otosclerosis. According to him, the essential factor for the development of the disease is stasis in the veins of the labyrinthine capsule with reversal of the direction of the blood stream, which leads to serious consequences, because there are practically no anastomoses between the vascular areas. It causes halisteresis of the bone of the labyrinthine capsule, which is followed by secondary changes in the halisteretic bone. The first stage of the otosclerotic focus is represented in hematoxylin-eosin slides by vivid red staining of the areas of the bone immediately surrounding the vessels. Cementing lines at the border between the diseased and the normal bone are not observed. At the same time the affected areas of the bone become swollen. This first stage is followed by partial resorption of the diseased bone. Later the areas of bone which have not been resorbed are impregnated again with calcium salts. This reimpregnation manifests itself in specimens stained with hematoxylin and eosin by violet or blue staining. The reimpregnated bone is later destroyed to a great extent by osteoclasts. Eventually new formation of red bone and retransformation of parts of the blue bone into red bone take place. The change from bone destruction to new formation of bone occurs as soon as the venous stasis which was responsible for all the described changes is eliminated. The elimination

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of the venous stasis is brought about by formation of new venous anastomoses with neighboring vascular areas, particularly with the vessels of the middle ear mucosa.

In pursuit of the idea that stasis with reversal of the direction of the blood stream within the veins of the labyrinthine capsule is the most important factor for the development of otosclerosis, Wittmaack tried to produce otosclerotic foci in the labyrinthine capsule of chickens. He produced stasis in the veins of the labyrinthine capsule and the adjacent areas and claimed to have produced changes which were histologically identical with those observed in human otosclerosis. The only difference in his opinion consisted in the fact that the changes in the experimental animals were equally distributed over the entire capsule whereas the changes in human otosclerosis are found in certain localized areas. This difference was explained by Wittmaack by differences in the vascular arrangement. In human beings the vessels enter or leave the capsule at certain circumscribed areas whereas in chickens entering and leaving vessels are found more equally distributed all over the capsule. Wittmaack's ideas about the histogenesis of otosclerosis are contrary to those of the majority of the authors. The main controversy is concerned with the early stages of the disease. Only a few authors, such as Eckert-Möbius, Döderlein, Lange and Guggenheim, have expressed the belief that the otosclerotic bone, at least in the early stages, is not newly formed but is represented by regressively changed old bone. Politzer, Siebenmann, Neumann, Mayer, Nager and Meyer, Weber, Greifenstein and others, however, have convincingly demonstrated that the otosclerotic bone in its entirety is newly formed and that the formation of the otosclerotic foci is invariably preceded by destruction of the old bone of the labyrinthine capsule.

In spite of the divergent opinions in the interpretation of the histologic picture of otosclerosis, the fact remains that Wittmaack's work on experimental chicken otosclerosis is of great theoretic interest. His first report came out in 1919 and was supplemented by later publications (1924, 1930, 1931, 1932, 1933, 1939) and by the work of Werner, which dealt with the development and structure of the chicken capsule. Wittmaack's findings started a vivid controversy. Mayer (1920, 1924, 1930, 1932) and Kamio questioned the results of Wittmaack. In the opinion of these authors the labyrinthine capsule of chickens has a structure entirely different from the human capsule and therefore is not a suitable object for experiments. The authors claimed that the findings of Wittmaack did not show the characteristics of otosclerosis but were normal changes occurring during the course of development and aging of the capsule or they were incidental pathologic changes.

According to Meyer (1933), the labyrinthine capsule of the chicken is not comparable to the human capsule either with regard to its development or with regard to its finer structure. A weblike fibril bone (*geflechtartiger Knochen* of the German authors) comparable to the periosteal and the endosteal bone of mammals is not found. The periosteal layer consists of parallel layers of fine fibril bone; an endosteal layer is hardly existent. The enchondral layer consists originally of fine fibril, "skeinlike" bone (*Straehnenknochen*—Meyer, 1928) with cartilage remnants. By way of osteoclastic resorption beginning along the enclosed vessels, an extensive reconstruction of the enchondral and of a part of the periosteal capsule takes place in the course of which the skeinlike bone and the cartilage remnants gradually and sometimes completely disappear. The resorption spaces are filled with "blue bone" which is poor in fibrils. The reconstruction continues during the entire life and leads to formation of a true breccia. This marked tendency of the chicken capsule toward internal reconstruction may, in Meyer's opinion, account for Wittmaack's results. In view of the essential biologic differences between the labyrinthine

capsule of chickens and that of human beings he questions that conclusions drawn from the experimental results could be applied to the human capsule. Wittmaack and Werner insisted that although there are certain differences in the histologic structure of the capsule they are by no means significant. The process of physiologic internal reconstruction is practically finished by the end of the ninth month.

Most of Wittmaack's experiments were performed on chickens of two years, and so a confusion of normal developmental with experimental changes was not possible. Otosclerotic changes were found in 85 to 90 per cent of the experimental animals. Similar spontaneous pathologic changes were found in only 10 per cent of the untreated chickens, so that in Wittmaack's opinion, also, a confusion with incidental pathologic changes was out of the question. Weber (1935) confirmed the histologic identity of the experimental changes with certain stages of human otosclerosis but he disagreed with Wittmaack about their histogenesis. In order to settle the controversy, I studied the development and histologic structure of the labyrinthine capsule in normal chickens and in chickens in which generalized osteodystrophia fibrosa was experimentally produced (Altmann, 1937). These investigations showed the following results.

In newborn chickens extensive areas are already ossified, and in the fifth postembryonal month the ossification is practically complete. The capsule consists of three layers, an enchondral, a thin endosteal and a periosteal layer. The enchondral layer consists of fine fibril skeinlike bone which shows in hematoxylin-eosin specimens a bluish stain and contains some but not many cartilage remnants. Contrary to the findings in the human capsule, an extensive internal reconstruction takes place in the enchondral layer of the chicken capsule throughout the last months of embryonic and the first six months of postembryonic life. Parts of the skeinlike bone and of the globuli ossei are destroyed and replaced by pinkish, fine fibril bone in which the fibrillae show a more parallel course (*parallelfaseriges Knochengewebe*—von Ebner). Due to this extensive internal reconstruction, the enchondral layer contains numerous cementing lines and lines of junction. The endosteal and periosteal layers consist also of fine fibril bone. The areas first formed show an irregular weblike disposal of the fibrillae and contain also preexistent connective tissue fibers. The areas which are formed later show a more parallel course of the fibrils.

This relatively simple picture becomes more complicated through the action of two factors, pneumatization, which attacks the capsule from outside, and growth changes, which attack the capsule from inside. Pneumatization starts soon after the onset of the ossification. It progresses either on a broad front from the outside toward the interior and may go as far as to the endosteal layer or penetrates into the enchondral layer and progresses in this layer along the longitudinal axis of the capsule parallel to its surface. The pneumatization goes on at a fast pace until near the end of the third month. Later it slows down considerably and is usually completed by the end of the first year, in some cases by the end of the second year. Histologically, the actual process of pneumatization is preceded by osteoclastic destruction of the bone either from the outer surface or along the vessels, with formation of perivascular resorption spaces filled with fine fibril cellular tissue. After the pneumatization has come to a standstill, apposition of parallel fibril bone to the walls of many of the pneumatic cells occurs, and in addition many of the perivascular spaces which preceded the pneumatization are filled up again with parallel fibril bone.

The changes due to the growth of the capsule take place during the first two months. They start from inside, from the lumen side of the labyrinth, and

consist in osteoclastic destruction of certain parts of the capsule together with apposition of newly formed parallel fibril bone. In confirmation of Wittmaack's observations it was found that the supplying blood vessels enter the capsule at many points and are not confined to certain areas as in the human capsule. At the end of the first year of life the labyrinthine capsule has reached its definite structure. Only in a few cases does pneumatization continue to the end of the second year or later.

Thus, during the early stages of its development the chicken capsule is similar in structure to the human capsule. The main differences consist in a more extensive and much longer lasting reconstruction of the enchondral capsule, with extensive replacement of the skeinlike bone by parallel fibril bone, and, in addition, in much more marked changes due to the pneumatization and the growth of the osseous capsule. Another difference lies in the fact that lamellar bone is not found in birds, its place being taken by the more primitive parallel fibril bone. The structural differences between the chicken capsule and the human capsule were not considered sufficient to preclude the use of chickens for experimental investigations.

In spite of the fact that the process of development and growth of the capsule is finished at the end of the first year, changes were also found in animals older than one year. These changes were considered, contrary to the opinion expressed by Mayer, Kamio and Meyer, as pathologic. In 3 of 10 animals perivascular resorption spaces were seen in the labyrinthine capsule and in the cortical layer of the bones of the skull. These spaces resembled the spaces seen in the course of pneumatization but frequently the tissue was richer in cellular elements. There was osteoclastic destruction in some parts of the osseous walls of these spaces with apposition of new parallel fibril bone in others. In 6 of the 10 cases other changes were observed in the labyrinthine capsule, the columella and the cortical layer of the bones of the skull. Spaces were seen which contained fine fibril, cellular bone marrow with engorged vessels and sometimes small accumulations of giant cells. Within this marrow there was new formation of blue weblike fibril bone which was poor in fibrils. This newly formed weblike fibril bone itself in many places underwent extensive destruction with new formation of other weblike fibril bone. Only in 1 case did the newly formed weblike fibril bone show a red color in specimens stained with hematoxylin and eosin. In this case it was richer in fibrils than usual.

Both of these changes were considered as manifestations of a generalized disease of the skeleton, as some kind of osteodystrophia fibrosa, possibly on the basis of a disturbance of the mineral metabolism. This assumption was supported by the fact that identical changes were found by Oberling and Guérin in the skeletons of chickens kept for nine to twelve months in cages without sand and by other authors in mammals kept in captivity on a special diet. Oberling and Guérin stated that it is well known among chicken breeders that chickens kept in wooden or metallic cages present after a certain time skeletal changes characterized by softening and deformation, particularly of the sternum, the femur and the tibia. In the opinion of the authors, in these chickens a disturbance of the mineral metabolism causes hyperfunction and hypertrophy of the parathyroid glands, which leads to mobilization of the calcium of the bones and eventually to chronic osteopathy. The whole process is reversible and stops when the primary metabolic disturbance disappears. The chickens examined by me were raised in captivity, on a chicken farm; no exact data were available concerning their food. It is easily conceivable, however, that food consisting mainly of corn with its very low calcium and high phosphorus content might lead to similar disturbances and similar skeletal changes. A comparison of my specimens with several of Wittmaack's specimens showed

that the histologic pictures of the second kind in my specimens were identical with Wittmaack's findings in so-called experimental otosclerosis. Furthermore, they were histologically identical with the blue-staining bone, and in 1 case with the early stage of red bone in human otosclerosis.

These investigations showed that histologic changes identical with those regarded by Wittmaack as experimentally produced were found in approximately the same percentage in untreated animals. But this does not necessarily mean that these changes are always present in this high percentage or at all in untreated animals and does not preclude the possibility that in certain cases identical changes might be produced by experimental venous stasis. It shows only that one has to select carefully the animals which are used in experimental work. A sufficient number of control animals should always be examined in order to exclude with certainty the presence of a generalized bone disease which might give exactly the same histologic picture as that expected as a result of the experiments. Up to the present time these indispensable precautions were taken neither by Wittmaack nor by his critics, and therefore the whole question of experimental otosclerosis remains undecided.

The examination of 11 chickens with generalized osteodystrophia fibrosa which histologically was similar to von Recklinghausen's form (according to Gohs, the disease was experimentally produced) showed that changes were also present in the labyrinthine capsule. But in these chickens they were much less marked than in the rest of the skeleton. These findings were regarded as an indication that the chicken capsule shows a greater tendency to internal reconstruction than the human capsule but that when the changes in it are compared with those in the rest of the skeleton, it exhibits the same relative behavior as the human capsule.

A few months later Oesterle (1937) discussed the development and structure of the chicken capsule. He obtained more or less the same results that I did but claimed that reconstruction processes in the enchondral layer go on throughout life. Perivascular resorption spaces are formed, and the original bone and many of the cartilage remnants are destroyed in these active areas. Then the resorption spaces are filled up with pink-staining parallel fibril bone. The bone originally formed shows a bluish stain and is homologous to the skeinlike bone in the human capsule, but the bundles of fibrils are thinner and show a more irregular course. Oesterle felt that it was difficult to decide whether these histologic differences were marked enough to make chickens unfit for experimental work on otosclerosis or not. Formation of blue bone poor in fibrils as described by Meyer and by me was not observed in his specimens, or at least it was not mentioned in his paper. Nevertheless, for some unexplained reason, he stated that the criteria regarded by Wittmaack as characteristic for otosclerosis—engorgement of vessels, blue staining of bone around the vessels and sievelike perforation *(siebförmige Durchlöcherung)* of the bone in the blue-stained areas—are normal features of the labyrinthine capsule in chickens.

In 1938 Meyer reiterated the opinion expressed in 1933:

> The extensive resorption in the region of the enchondral capsule in the shape of osteoclastic widening of the enclosed vascular channels with new formation of blue bone which itself, by means of repeated reconstruction, may eventually become a breccia, was regarded by Kamio, Mayer, Meyer and Oesterle as physiologic internal reconstruction, but recently by Altmann as a spontaneous pathologic process, as osteodystrophia fibrosa. . . .

He apparently overlooked the fact that Oesterle did not describe new formation of blue bone but only new formation of pink parallel fibril bone. Meyer conceded that the changes leading to the new formation of blue bone as well as the changes

seen by Wittmaack in his experimental animals were similar to those found in human osteodystrophia fibrosa, but he was not sure that they actually were identical. "But whether this similarity is only superficial, due to similar staining of the bone in hematoxylin-eosin specimens, seems doubtful in spite of Altmann's claim that he found true weblike fibril bone in the resorption spaces. Oesterle, on the other hand, did not find this type of bone tissue." Meyer apparently did not realize that two kinds of bone are found in the resorption spaces. The pink-staining parallel fibril bone which is regarded by me as a normal occurrence in the course of internal reconstruction and which also is present in a certain type of generalized osteodystrophia fibrosa, but which Oesterle stated is always a normal finding in the course of internal reconstruction. The blue bone described by Oesterle is the primarily formed bone of the enchondral capsule and has nothing to do with the blue bone secondarily formed in resorption spaces as described by Meyer and by me. Therefore, it seems difficult to understand why Meyer held that Oesterle's findings disprove my statement that the secondarily formed blue bone shows the structure of weblike fibril bone.

Meyer expressed the opinion that a final decision as to whether the new formation of blue bone is normal or pathologic is at the present time impossible. The normal bone of birds has a structure different from that in mammals. In his opinion it remains to be seen whether these two kinds of bone tissue react identically or differently under the same pathologic conditions. In addition he said that it is well conceivable that reactions which appear pathologic in mammals might be found normal in birds. In conclusion he stated that according to the present state of knowledge there are five possibilities in explanation of the controversial processes in the chicken capsule. 1. They are normal processes of internal reconstruction which are morphologically much like those found in osteodystrophia fibrosa of man and some laboratory animals (Kamio; Mayer; Meyer; Oesterle). Special studies on the entire chicken skeleton should be done in order to elucidate this question. 2. In view of the particular arrangement of the vessels and other structures and the particular biology of the chicken capsule, these normal processes might be increased by venostasis (Meyer). 3. They are manifestations of generalized osteodystrophia fibrosa (Altmann). 4. These changes due to generalized osteodystrophia fibrosa might be increased by venostasis. 5. They are true otosclerotic changes of the capsule due to venostasis, in rare cases spontaneous otosclerotic changes (Wittmaack). There is still no proof for this assumption due to the lack of investigations concerning general questions of the physiologic internal reconstruction of the bones in chickens and concerning generalized osteodystrophia fibrosa. Nager (1939) asserted that "it is now generally accepted that the alterations by experimental venous stasis in fowls—if they are not due to the normal development—are not identical with human otosclerosis; at any rate they could not be produced in monkeys." When I published my paper (1937) I thought that the facts put forward in support of the opinion that the changes ocurring in the chicken capsule after the first year are pathologic, and not normal, were rather conclusive. Meyer's critique of my results shows that I was mistaken in this assumption. In order to settle this matter new evidence had to be collected. Studies concerning the physiologic internal reconstruction of the bones in birds seemed to be difficult. In order to obtain chickens of exactly known age one has to take animals raised in captivity. But in this case a strong possibility exists that the animals suffer from a generalized disease of the skeleton. Various and complicated precautions have to be taken in order to avoid this complication. As to wild birds it could be assumed with a fair degree of probability that they are more or less free from the more serious pathologic bone affections resulting from factors

connected with domestication. These animals can choose their food and their living conditions to a great extent. But an exact determination of their age, which is indispensable for the study of the physiologic internal reconstruction of the bone, is practically impossible. Under these circumstances another approach to the problem was preferred. Studies of the structure of the labyrinthine capsules of several kinds of wild birds were undertaken. If the changes in question are really physiologic and occur at all ages, as Mayer, Kamio and Meyer claimed, one should expect to find them not only in chickens but also in wild birds. Several species of wild birds were examined (ruffed grouse, American quails, ring-necked pheasants, American woodcocks, wild ducks) in order to refute a possible objection that findings in one particular species might be due to inherent differences between the species in question and chickens.

The material for these investigations was supplied by Dr. John D. Kernan, director of the department of otolaryngology. The investigations were carried through with the support of a grant from the Research Council of the American Otological Society.

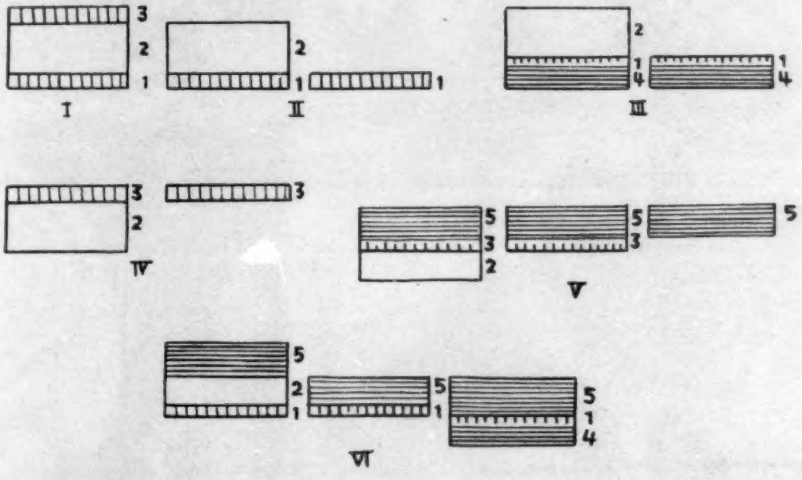

Fig. 1.—Schema of the more frequent variations in the structure of the labyrinthine capsule.
I. Original three layers; *1,* endosteal layer; *2,* enchondral layer; *3,* periosteal layer.
II. Erosion from the outer surface.
III. Erosion from the outer surface with apposition of parallel fibril bone (*4*) to the inner surface.
IV. Erosion from the inner surface.
V. Erosion from the inner surface with apposition of parallel fibril bone (*5*) to the outer surface.
VI. Apposition of parallel fibril bone to the outside after erosion from the outside and in one instance apposition to the inside and outside after resorption from both sides.

HISTOLOGIC FINDINGS IN THE SKULLS OF SEVERAL TYPES OF WILD BIRDS

Ruffed Grouse (Bonasa umbellus).—Eleven wild living birds were examined. All apparently were adults and showed complete ossification and full pneumatization of the skull. Only in some places at the periphery new formation of small air cells was noticed. The structure of the labyrinthine capsule is essentially the same as in chickens. Because of the fact that only adults were examined, the three layers of the original anlage (endosteal, enchondral and periosteal) are nowhere seen any more. The labyrinthine walls show the same structural variations as in adult chickens; they are well demonstrated in a schema taken from my previous paper

(1937) (fig. 1). The enchondral layer consists mainly of small pieces of bluish-reddish skeinlike bone, separated from each other by cementing lines and lines of junction. It contains relatively numerous cartilage remnants, particularly in

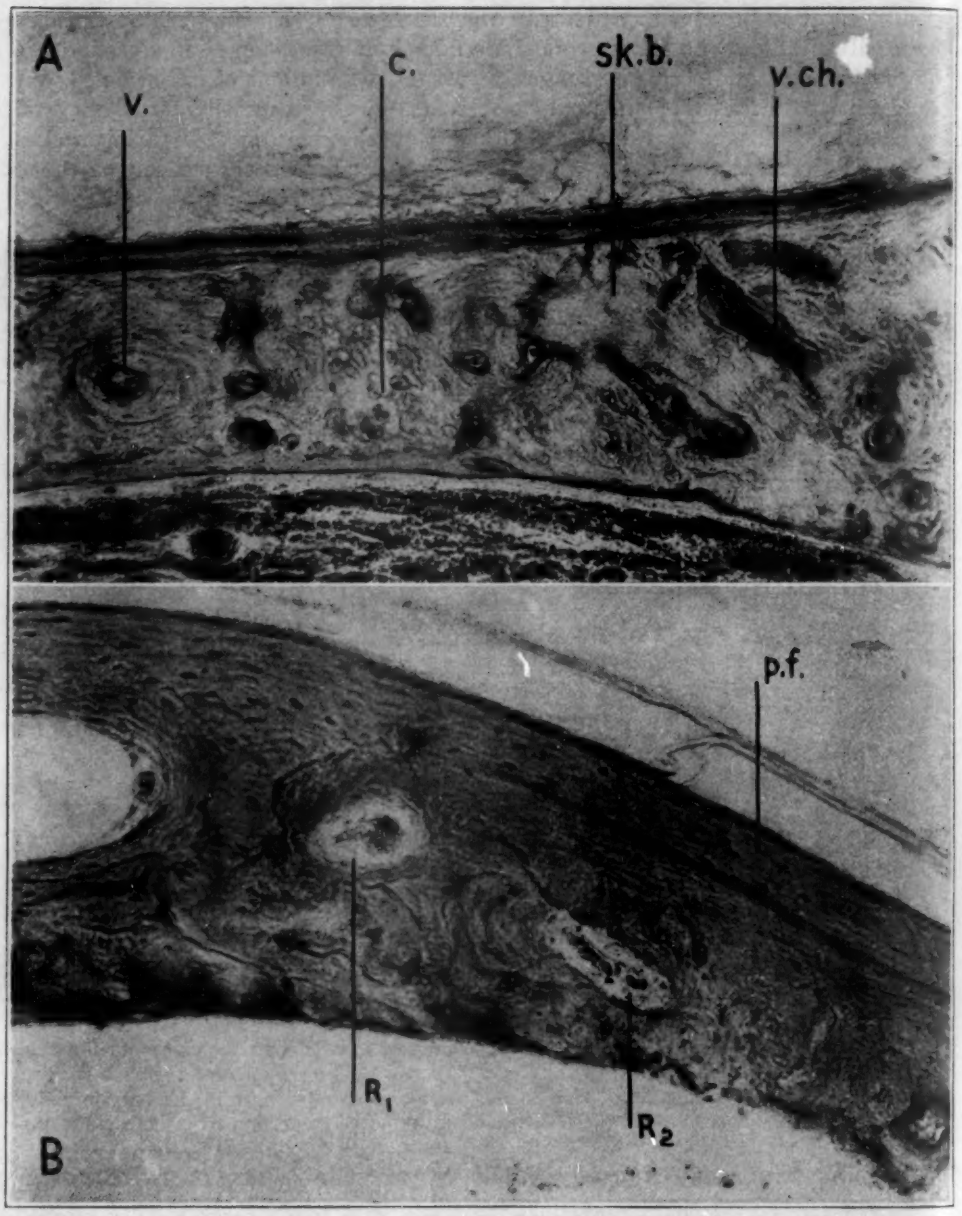

Fig. 2.—*A* (ruffed grouse 2, section 410), structure of the enchondral capsule: *c*, cartilage remnant; *sk.b.*, skeinlike bone; *v.*, vessel surrounded by parallel fibril bone; *v.ch.*, vascular channel with heavily blue-stained sheath *(Grenzscheide)*.

B (ruffed grouse 9, section 120), two resorption spaces (R_1, R_2) in the enchondral capsule, one filled with parallel fibril bone (R_1). Note the outer layer of parallel fibril bone (*p.f.*).

the medial wall of the vestibule and cochlea. There are relatively few areas of pink-staining parallel fibril bone surrounding a central vessel and separated from the

adjacent bone by a cementing line. The areas represent bone which was newly formed in the course of the internal reconstruction of the capsule (fig. 2*A*). Only in 1 capsule (fig. 2*B*) are a few small resorption spaces seen, two in the wall of a semicircular canal and one in the medial wall of the cochlea. Each of these spaces contains a small vessel in the center and is filled with a fine fibril connective

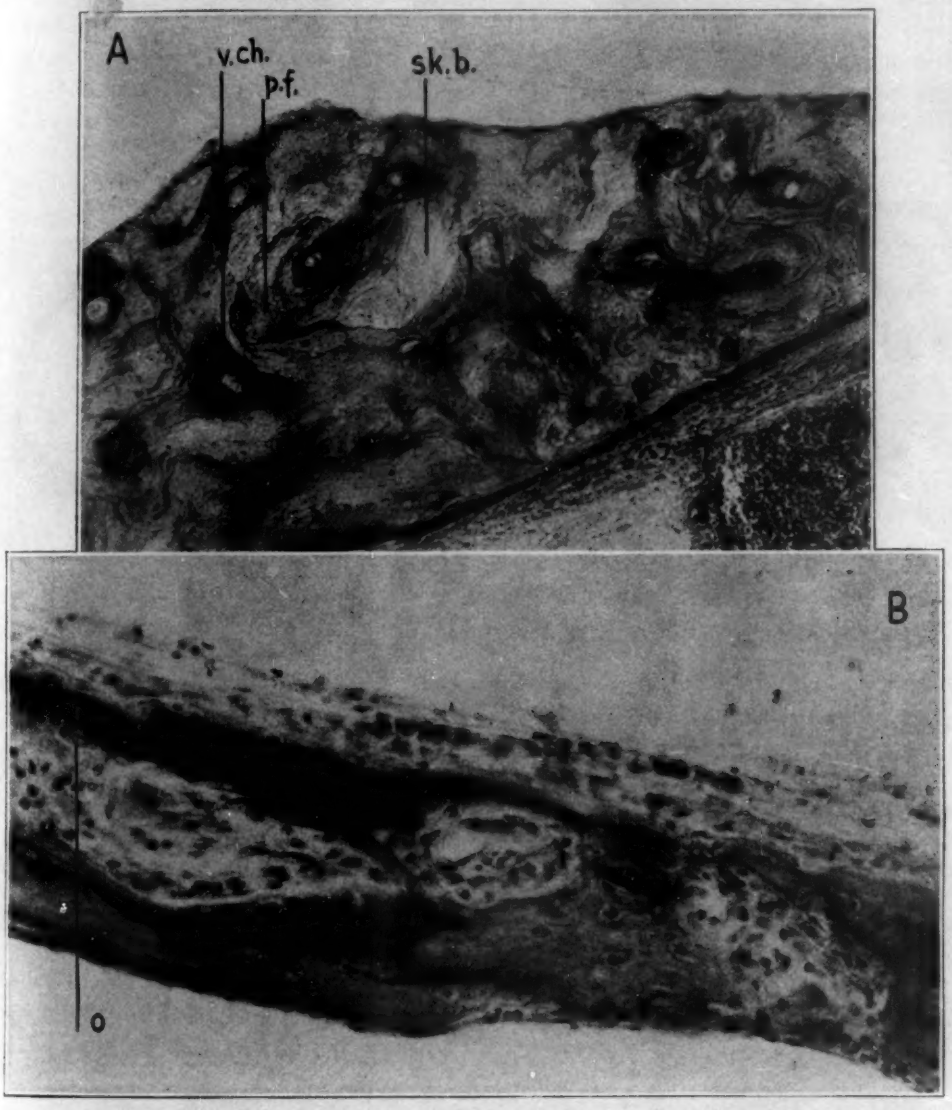

Fig. 3.—*A* (American quail 6, section 250), normal structure of the enchondral capsule: *p.f.*, parallel fibril bone; *sk.b.*, skeinlike bone; *v.ch.*, vascular channel with heavily blue-stained sheath.

B (American quail 2, section 420), internal reconstruction of the capsule from perivascular resorption spaces. Note osteoclast (*o*).

tissue which is moderately rich in cells. In the wall of one space, at its circumference there is beginning new formation of bone; otherwise there is neither new formation nor destruction. The blood vessels show the same arrangement as in the chicken

capsule, and the marginal sheaths of the vascular channels (*Grenzscheiden der Gefässkanäle*) show the same deep blue stain. In none of the specimens are areas of newly formed blue weblike fiber bone found. The endosteal layer is poorly

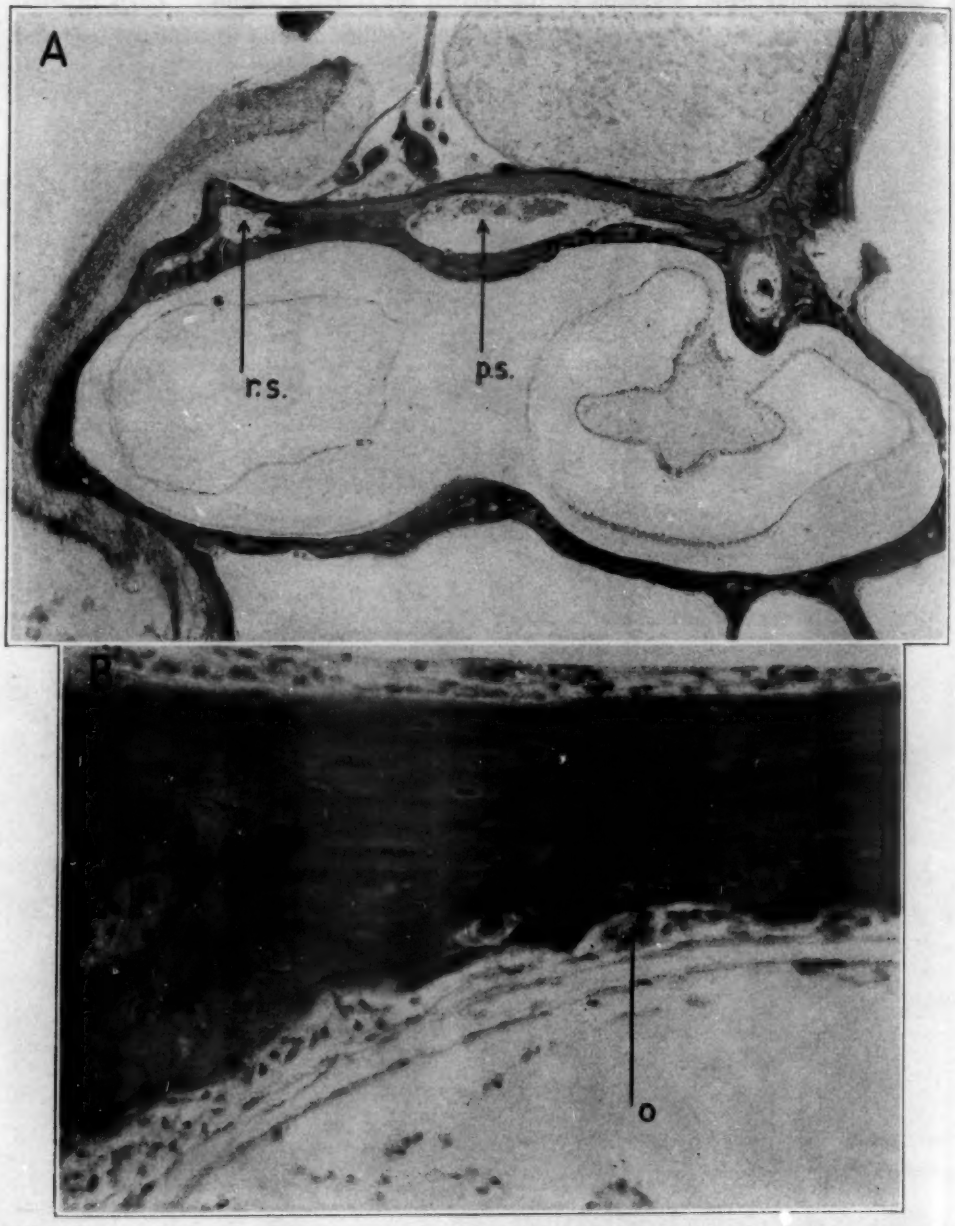

Fig. 4.—*A* (American quail 2, section 450). "longitudinal" pneumatization of the medial wall of the capsule: *p.s.*, pneumatic space; *r.s.*, resorption space preceding the pneumatization.

B (American quail 2, section 420), eccentric growth of a semicircular canal with osteoclastic erosion of the inner surface. Note osteoclast (*o*).

developed. The original periosteal layer has been separated from the rest of the labyrinthine capsule or destroyed in the course of pneumatization. In many places a layer of parallel fibril bone of varying thickness has been deposited at the

outer surface of the enchondral capsule, and this parallel fibril bone represents the analogue but not the homologue of the periosteal layer of the mammalian capsule. Compared with the chicken capsule the grouse capsule shows evidence of a lesser degree of internal reconstruction. This is indicated by the presence of more numerous cartilage remnants and fewer areas of parallel fibril bone, for the parallel fibril bone is formed after destruction of the original skeinlike bone and of the cartilage remnants in the same areas.

American Quail (Colinus virginianus).—Fourteen birds were examined. Twelve of them apparently were adults; their skulls were completely ossified and pneumatized; only in some peripheral areas could a new formation of a few small cells be observed. The structure of the labyrinthine capsule was practically identical with that of the chicken capsule (fig. 3*A*). In 9 of the 12 animals the process of internal reconstruction had come to a complete standstill. In three some resorption spaces were seen in the medial wall of the vestibule and some in adjacent areas of the semicircular canals. The spaces were filled with fine fibril marrow and the walls contained in a few places osteoclasts in Howship's lacunas or a thin layer of newly formed osteoid. In 2 of these cases some resorption spaces of similar structure were also seen in other parts of the skull. One quail was a young animal. The ossification of the skull and the pneumatization were still in progress. The labyrinthine capsule showed the internal reconstruction, pneumatization and growth changes in the bone of the semicircular canals still going on. The changes were identical with those observed in the chicken capsule (figs. 3*B* and 4). In 1 quail the skull was already completely ossified but pneumatization was still in progress at the calvarium and at the base of the skull. This animal evidently was relatively young. In this case a few resorption spaces were found in the medial wall of the vestibule and cochlea and in the semicircular canals. Similar spaces were present at the base of the skull. Compared with that of the grouse capsule the enchondral layer contained less numerous cartilage remnants and more areas of parallel fibril bone. In other words, there was evidence that more extensive internal reconstruction had taken place than in the grouse.

Ring-Necked Pheasant (Phasianus torquatus).—Ten birds were examined. In 7 of them the pneumatization of the labyrinthine capsule was at a complete standstill. The capsule contained few cartilage remnants, indicating that extensive internal reconstruction had taken place; but neither in the labyrinthine capsule nor in the rest of the skull were resorption spaces visible. In 1 animal there was little progress of the pneumatization in the peripheral parts of the skull but the labyrinthine capsule contained somewhat more numerous cartilage remnants and showed signs of a moderate degree of internal reconstruction. In 1 animal the pneumatization was still progressive in the skull and in the labyrinthine capsule. The capsule itself contained numerous cartilage islands and showed more marked signs of internal reconstruction. One animal was evidently young. It showed markedly progressive pneumatization of the skull and of the labyrinthine capsule. The capsule contained numerous and relatively large cartilage islands and many perivascular resorption spaces. Some of the cartilage remnants showed destruction by chondroclasts; in other resorption spaces the walls were lined with a layer of newly formed osteoid.

American Woodcock (Rubicola minor).—Two birds were examined. Both were adult animals. The pneumatization of the labyrinthine capsules was at a complete standstill. They contained few cartilage remnants. There were no signs of internal reconstruction and no resorption spaces in the rest of either skull.

Wild Duck (Aix sponsa).—Two birds were examined. Both were adult animals. The pneumatization of the labyrinthine capsules was complete. They contained few cartilage remnants. One capsule contained no resorption spaces; the other one and also some other bones of the skull, a few spaces. In the majority of these spaces the walls were covered with a thin layer of osteoid; in some places osteoclasts in Howship's lacunas were visible.

Comment.—The examination of the labyrinthine capsules of several types of wild birds shows that resorption spaces are rare in adult birds, in which pneumatization had already come to a complete standstill. They were found five times among 34 birds but were in no instance numerous. Three times they were seen in the capsule as well as in the skull, twice only in the capsule. The interpretation of these resorption spaces is not easy. They show the same histologic picture as those observed in the course of pneumatization. At the time when the pneumatization comes to a standstill, the pneumatic cells which have penetrated most deeply into the capsule are surrounded by moderately numerous resorption spaces. These spaces are gradually filled up by apposition of parallel fibril bone to the walls. A further complication is caused by the fact that they can hardly be distinguished from the resorption spaces which occur in the first stages of certain generalized diseases of the skeleton (Altmann). Bremer came to similar conclusions and stated that the microscopic picture seen in the expanding zone of the active bone changes beyond the limit of the entering air sac reminds one strongly of similar pictures seen in mammalian bones which have been altered by the disease called osteitis fibrosa. The cases in which the pneumatization was already at a complete standstill and in which only a few resorption spaces were seen in the capsule, with none in the rest of the skull, could be explained as due to prolonged internal reconstruction. This assumption seems possible particularly in regard to animals in which the internal reconstruction is always intensive, e. g. in quails, but a final decision is hardly possible. The cases in which other parts of the skull in addition to the capsule were affected are more likely to be caused by a very mild degree of a generalized disease of the bone. In none of the wild birds was any new formation of bluish-reddish weblike fibril bone observed, although such new formation was found by me in the majority of the adult chickens and was regarded by me as a pathologic finding, as a manifestation of generalized osteodystrophia fibrosa, possibly due to a metabolic disturbance.

In order to determine whether this assumption was correct or not, I attempted to produce similar bone changes experimentally by putting the birds on a diet rich in phosphorus and poor in calcium. Five quails (a sixth served as a control animal) and 6 pheasants were fed with three parts of corn and one part of wheat. Twice a week they got in addition a few green salad leaves. The birds were kept in cages made from wire and had sufficient daylight.

HISTOLOGIC FINDINGS IN THE SKULLS AND SKELETONS OF THE EXPERIMENTAL ANIMALS

American Quails (Colinus virginianus).—The labyrinthine capsule of the control animal, killed at the time of the onset of the experiment, showed the same histologic picture as did that of the wild birds. The skull was completely ossified but the pneumatization was still progressive in the periphery. There was also new formation of some pneumatic cells in the medial wall of the vestibule. Other resorption spaces were completely absent in the capsule as well as in the other parts of the skull. Among the 5 experimental animals 1 died after seven and a

half and 1 after eight months, while 3 survived and were killed after nine months. In addition to the skulls, the sternum, the tibia and the femur and the parathyroid glands were examined. The histologic examination of all the animals showed evidence of a disease of the labyrinthine capsule which in all but 1 was combined with a generalized disease of the skeleton. Though the animals were kept under identical conditions, the extent of the changes showed great variations. In all of them the pneumatization had come to a complete standstill.

One animal (D 7), killed after nine months, showed in the capsule of the concavity of the semicircular canals, which is always much thicker than that of

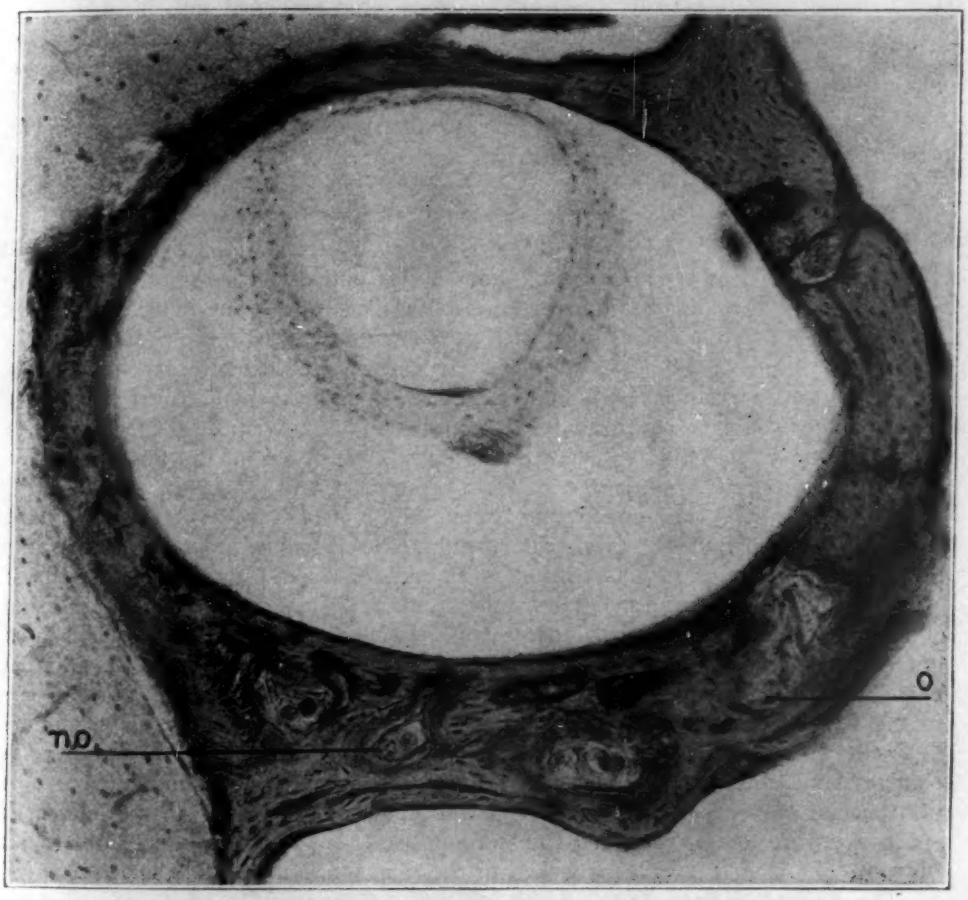

Fig. 5 (American quail D 6, section 170).—Several perivascular resorption spaces in the capsule of the concavity of a semicircular canal. Note apposition of osteoid covered with osteoblasts (*n.o.*) to the wall of a resorption space and an osteoclast in the wall of a resorption space (*o*).

the convexity, a few perivascular resorption spaces, filled with a fine fibril, rather cellular bone marrow. There was neither new formation of osteoid nor osteoclastic destruction in the walls. The rest of the skull and of the skeleton was free from change.

The second animal (D 6), killed after nine months, showed throughout the capsule numerous perivascular resorption spaces containing hyperemic vessels and fine fibril, cellular marrow. There were only a few osteoclasts in the walls, and

in some of the spaces the walls showed a thin layer of osteoid which was covered with osteoblasts. The resorption spaces were much more numerous in the thicker than in the thinner parts of the capsule (fig. 5). In some circumscribed areas of the semicircular canals the inner layers of the capsule or even the entire capsule was destroyed and replaced by a network of newly formed bone. This bone took a more bluish stain and showed in many places a small marginal layer of osteoid which was covered by rather flat osteoblasts. On examination with polarized light this bone was found to be a weblike fibril bone which was relatively poor in fibrils. There was always a sharp separation between the old and the newly formed bone. In some areas the newly formed bone extended into the lumen of the semicircular canals and caused a slight narrowing of the perilymphatic space. Resorption spaces were also present in the two uppermost vertebrae; the rest of the skull was free from changes. The sternum contained a few resorption spaces with progressive osteoclastic destruction in some of them and new formation of osteoid in others. Within the central marrow space of the tibia, new formation of a network of weblike fibril bone was seen in a circumscribed area. The femur was free from changes.

The third animal (D 3), which died spontaneously seven and one half months after the onset of the experiment, showed the entire old labyrinthine capsule replaced by a network of bluish-staining weblike fibril bone which contained relatively wide marrow spaces filled with the same marrow as in the preceding cases (fig. 6 *A*). Many of the newly formed trabeculae showed osteoid margins; others, evidence of osteoclastic destruction. The newly formed bone filled the entire perilymphatic space and surrounded the membranous labyrinth. The growing of bone into the perilymphatic spaces had occurred mainly from the areas in which the changes were most marked, i. e., from the thicker and more vascularized areas of the capsule. In the semicircular canals these areas are represented by the concavity and the lateral parts. The membranous canal lies always eccentrically toward the convexity. This position is maintained even when the perilymphatic space is completely obliterated (fig. 6 *B*). Outward growing of the newly formed bone into the adjacent marrow spaces was nowhere observed. The changes were not confined to the capsule but extended for a short distance into the adjacent parts of the bones of the skull. The columella showed a few resorption spaces in the footplate which had been filled with newly formed osteoid. The annular ligament was intact (fig. 6 *A*). The rest of the skull showed in the thicker parts changes similar to those in the labyrinthine capsule and in the thinner parts only perivascular resorption spaces. Most of the thin trabeculae between the pneumatic spaces were free from changes. The sternum presented a picture of osteodystrophia fibrosa. The change was less marked in the tibia and still less in the femur.

The fourth animal (D 4), which died spontaneously eight months after the initiation of the experiment, showed extensive and active changes. The capsule was less uniformly affected than in the preceding case; some parts of the original capsule were still preserved, but they underwent rapid osteoclastic destruction from numerous perivascular resorption spaces. At the periphery of these areas large resorption spaces were present, filled with highly cellular, fine fibril marrow in which new formation of a network of bluish-reddish weblike fibril bone took place (fig. 7 *A*). This newly formed bone itself underwent a vivid reconstruction; it was being destroyed by osteoclasts and replaced by new weblike fibril bone. Eventually a network of slightly more reddish weblike fibril bone was formed which showed only few signs of reconstruction. Large areas of the capsule consisted entirely of this type of bone. The perilymphatic space was, as before described,

filled with newly formed bone. The latter was particularly extensive in the anterior vertical canal and had caused compression and collapse of the membranous canal, the lumen of which was slit shaped or completely obliterated. In some places the wall was folded and invaginated into the lumen (fig. 7 *B*). In several

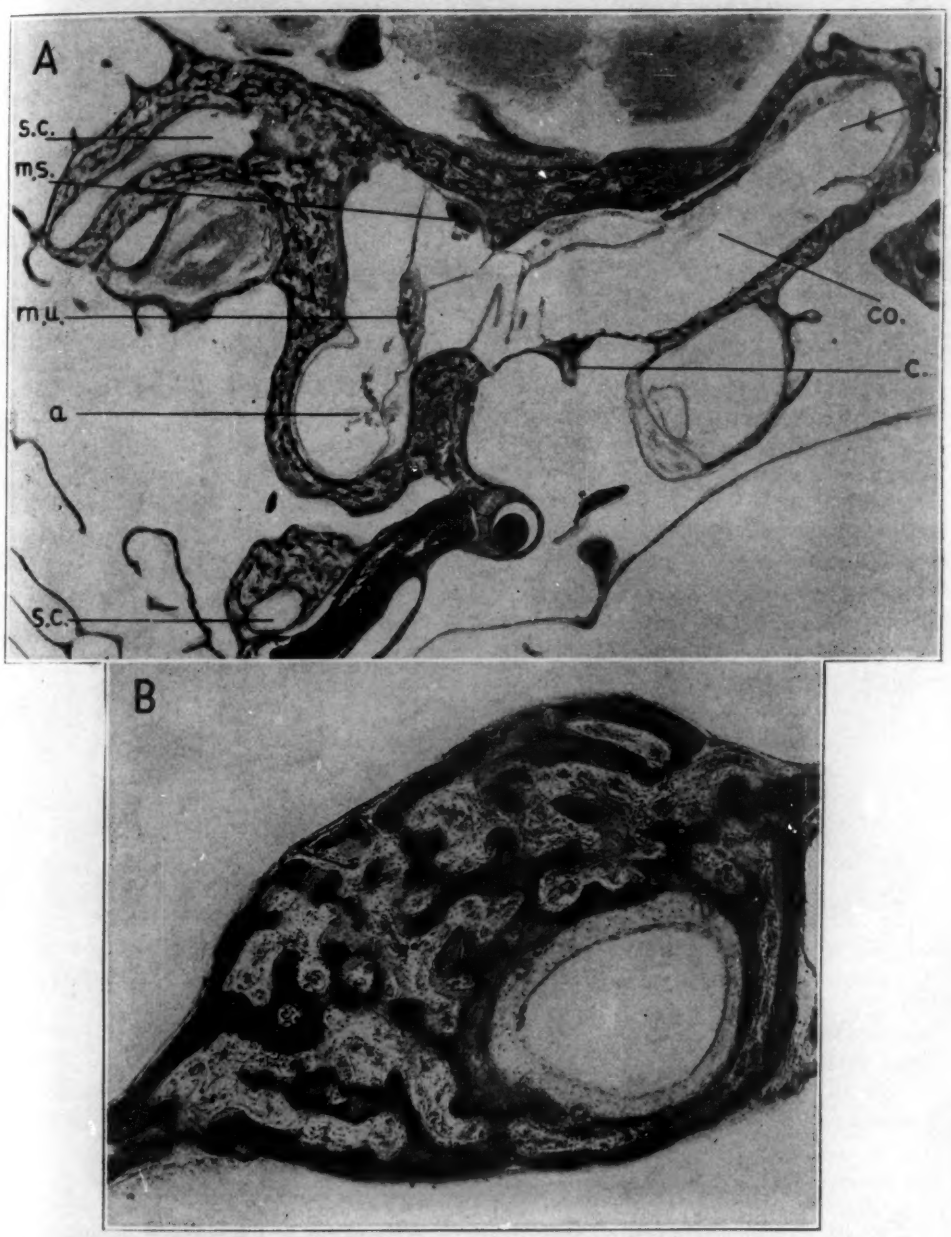

Fig. 6.—*A* (American quail D 3, section 230), replacement of the entire old labyrinthine capsule by newly formed weblike bone: *a*, ampulla of external semicircular canal; *c*, columella; *co*, cochlea; *l*, lagena; *m.s.*, macula sacculi; *s.c.*, semicircular canals; *m.u.*, macula urticuli.

B (American quail D 3, section 230), replacement of old bone of the capsule of a semicircular canal by newly formed weblike fibril bone; the perilymphatic space is completely obliterated.

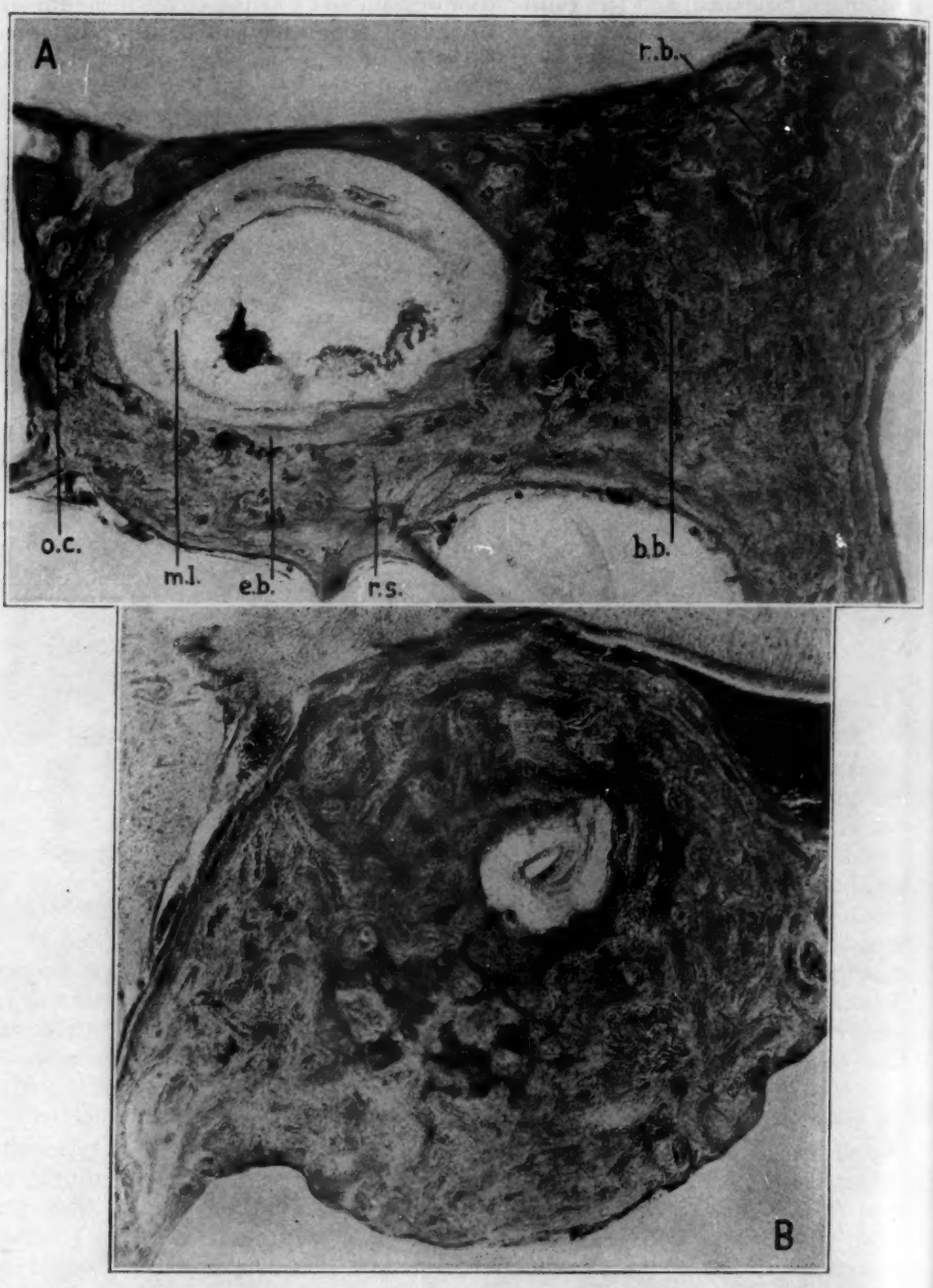

Fig. 7.—*A* (American quail D 4, section 270), replacement of the old labyrinthine capsule by newly formed weblike fibril bone: *b.b.*, bluish-staining weblike fibril bone with vivid internal reconstruction; *e.b.*, endosteal bone being eroded from the outside; *o.c.*, old capsule; *m.l.*, macula lagenae; *r.b.*, more reddish-staining weblike fibril bone with few signs of internal reconstruction; *r.s.*, large resorption spaces with new formation of weblike fibril bone.

B (American quail D 4, section 200), membranous anterior vertical canal completely surrounded and compressed by newly formed weblike fibril bone. Its wall is folded and invaginated into the lumen.

places the growing of the newly formed bluish-reddish bone into the perilymphatic space was preceded by new formation of pink-staining bone in the endosteum which protruded into the lumen. This bone was being eroded on its outer surface by osteoclasts and gradually replaced by the bluish-reddish bone (fig. 7*A*). In a very few places the newly formed bone grew from the outer surface of the capsule into adjacent pneumatic spaces, but this growth was nowhere strongly indicated. The columella contained some resorption spaces; on one side the upper part of the original footplate had been replaced by newly formed pink-stained parallel fibril bone. The annular ligament was intact on both sides. The changes described continued from the capsule for a short distance into the adjacent bones of the skull; the newly formed bone was separated from the old bone by cementing lines (fig. 8). The rest of the skull changed with the thickness of its respective bones. The thick bones showed identical changes; the thinner ones, only resorption

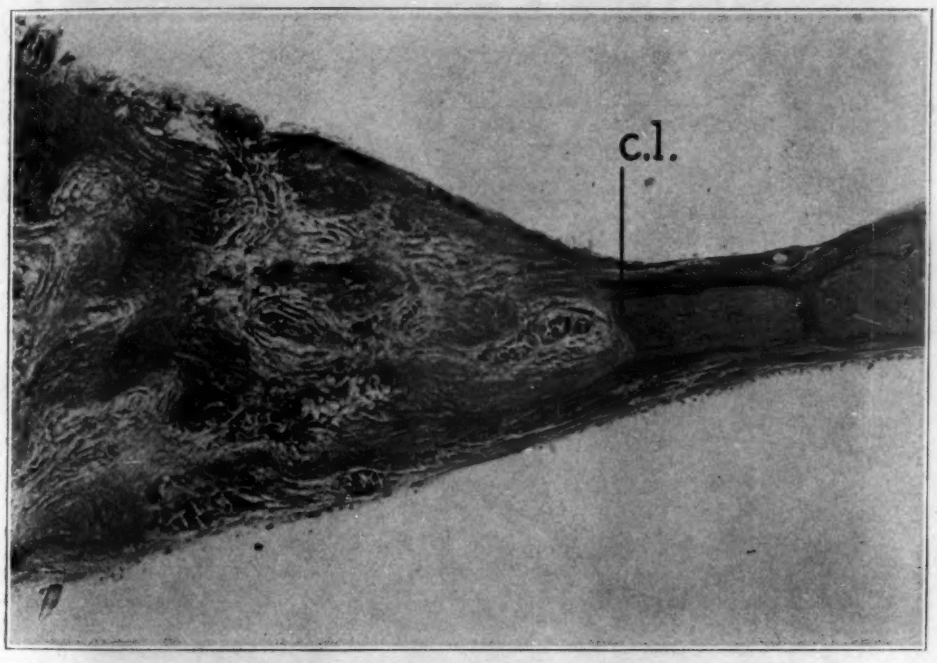

Fig. 8 (American quail D 4, section 230).—Newly formed bone separated from the old bone by a cementing line (*c.l.*).

spaces with osteoclasts and new formation of parallel fibril bone. The thin trabeculae between the cells were usually unaffected, but occasionally they showed some resorption spaces or even foci of newly formed weblike fibril bone in netlike arrangement. The sternum and the tibia showed a marked degree and the femur a somewhat less marked degree of osteitis fibrosa.

The fifth animal (D 5), which was killed nine months after the onset of the experiment, showed a very similar picture. Almost the entire old capsule had been destroyed, and there was less evidence of actual reconstruction in the newly formed bone present. The sternum showed the typical picture of osteodystrophia fibrosa, in some places with formation of small giant cell tumors. The changes in the tibia were somewhat less marked. The femur contained a few resorption spaces but was otherwise free from changes.

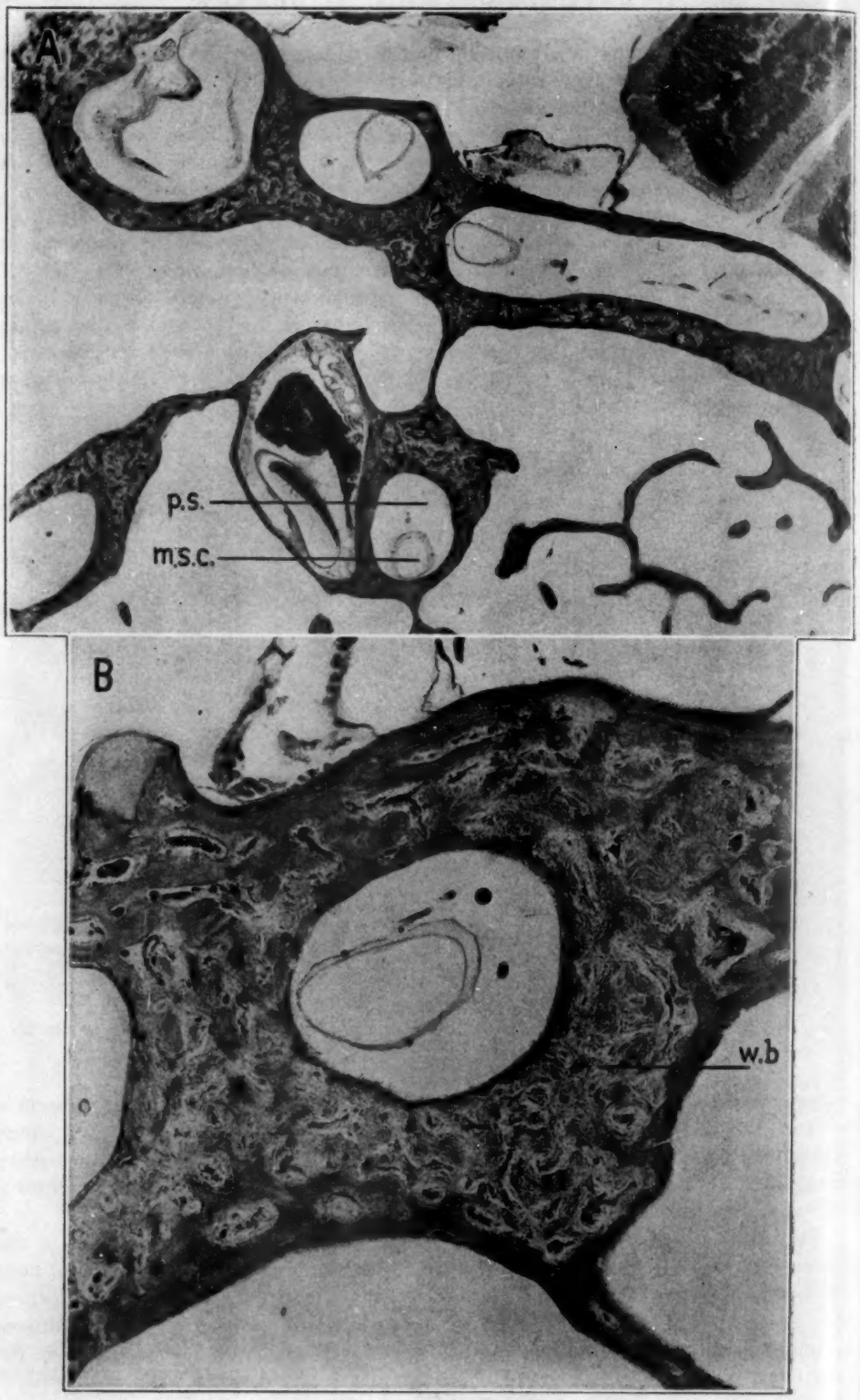

Figure 9

(See legend on opposite page)

The parathyroid glands were markedly enlarged in all the experimental animals. Their average volume was about six times larger than that of the control. The enlarged glands were surrounded by a greatly thickened fibrous capsule. There was considerable hypertrophy as well as hyperplasia of the glandular cells.

Ring-Necked Pheasants (Phasianus torquatus).—Two of the 6 experimental birds died spontaneously. One died one and one-half, and the other four months, after the onset of the experiments; the remaining 4 were killed after nine months. In addition to the skull, the sternum, the femur and the tibia were examined. In the animal which died after one and one-half months the pneumatization of the labyrinthine capsule was still progressing to some extent, and the capsule contained relatively numerous cartilage remnants and a few perivascular resorption spaces filled with a moderately cellular, fine fibril marrow and some osteoblasts in the walls. The sternum, femur, tibia and fibula were free from pathologic changes. In all the other animals pneumatization was at a complete standstill and the cartilage remnants were less numerous. All the pheasants killed after nine months showed resorption spaces in the capsule with osteoclasts in the walls but no evidence of new-formed bone. The spaces were scarce in 1, fairly numerous in 1 and very numerous in 2. In the latter 2 animals the capsule showed large areas of outspoken porosis. In the semicircular canals the changes were most marked in the convexity and least marked in the concavity. Similar resorption spaces were also found in the thicker parts of the other bones of the skull. Resorption spaces were also present in the sternum, the femur, the tibia and the fibula. The central marrow cavity of the tibia showed some fibrosis in the peripheral parts with new formation of weblike fibril bone in netlike arrangement. The pheasant which died after four months (D 2) showed a different picture. There was extensive progressive osteoclastic destruction of the old capsule by numerous perivascular resorption spaces, frequently with confluence of the spaces; in many of these spaces new formation of weblike fibril bone was taking place. The newly formed bone showed a more bluish stain than the bone of the old capsule and formed a fine network. In many places this newly formed bone was being destroyed by osteoclasts and replaced by later formations of weblike bone. Large parts of the old capsule were already completely replaced by newly formed bone but, as a rule, rests of the external cortex still persisted. The changes were particularly marked in the anterior vertical canal, and here the perilymphatic space was somewhat narrowed by the excessive new formation of bone in the concavity and the lateral parts of the osseous canal. The convexity of the canal wall showed only perivascular resorption spaces. The rest of the skull with exception of the thin intercellular septums contained numerous resorption spaces with progressive osteoclastic destruction but without any evidence of new formation of bone. The cortex of the tibia and that of the fibula contained numerous perivascular resorption spaces with osteoclasts in the walls and with new formation of osteoid in some places. The central marrow cavity was free from changes. Similar spaces were present in the femur but were less numerous. The sternum was almost free from changes.

EXPLANATION OF FIGURE 9

A (pheasant D 6, section 360), progressive porosis of the labyrinthine capsule as a result of the formation of numerous resorption spaces; *m.s.c.*, membranous semicircular canal; *p.s.*, perilymphatic space.

B (pheasant 2, section 210), new formation of weblike fibril bone (*w.b.*) in the wall of a semicircular canal.

Comment.—The histologic observations in quails and pheasants were identical with those of Oberling and Guérin in chickens. When put on a diet rich in phosphorus and poor in calcium and kept in cages without gravel, both kinds of birds after several months have a generalized disease of the skeleton, osteodystrophia fibrosa, which histologically shows similarities to osteitis fibrosa von Recklinghausen in man, and is identical with certain experimental changes of the skeleton in mammals (so-called "low calcium rickets"). It is not my purpose in this paper to discuss the mechanism by which the changes were brought about. I shall, however, mention that the parathyroid glands were considerably enlarged; in quails their volume was increased six times. The fact that such changes can be produced only by faulty nutrition should be emphasized.

The changes show two different degrees of intensity. In the mild degree, which was present in 1 quail and in 5 pheasants, there is osteoclastic destruction of the old capsule from perivascular resorption spaces, which may or may not be followed by or combined with apposition of new bone to the walls of the spaces. In the severe degree, which was found in 4 quails but in only 1 pheasant, the old capsule is in process of being completely destroyed and replaced by newly formed weblike fibril bone in netlike arrangement. This bone is relatively poor in fibrils and shows the same structure as the so-called blue bone in human otosclerosis. In many places it has undergone internal reconstruction and is in process of being destroyed by osteoclasts and replaced by another generation of weblike fibril bone.

There are marked variations not only in the intensity of the changes but in the length of time in which the changes develop in animals of different species kept under absolutely identical conditions and also in animals of the same species. Similar observations have been made by Oberling and Guérin in chickens and by other authors in experimental bone diseases in other animals. These observations prove that constitutional or other, still unknown factors play an important role in bringing about the changes. The lesser degree of intensity of the changes in pheasants could possibly be due to these factors, and it is conceivable that the changes would have become more marked if the animals had lived longer.

The marked narrowing or even complete obliteration of the perilymphatic spaces in cases with far progressed changes has never been observed before. It is due to two factors. When in the case of osteitis fibrosa an area of compact bone is replaced by a more or less loose network of newly formed bone, the affected area frequently increases in thickness because frequently the volume of the newly formed bone is larger than that of the old bone. In long bones of human beings an increase in the volume of the cortex may lead either to concentric thickening of the bone with narrowing of the central marrow cavity or to eccentric thickening of the bone without narrowing or to a combination of both processes. In the experimental animals there was almost exclusively concentric thickening of the labyrinthine capsules with subsequent narrowing or obliteration of the perilymphatic spaces. In addition, in several specimens new formation of bone within the endosteum was seen in circumscribed areas of the vestibule, of the semicircular canals and of the cochlea.

Concentric thickening of the labyrinthine capsule has never before been described. New formation of bone within the endosteum protruding into the perilymphatic space has been described as a rare occurrence in otosclerosis by Nager and Fraser. In some places the otosclerotic bone had penetrated bony masses formed in the endosteum. The two authors stated that such bone formation in the inner ear has been found only in otosclerosis, whereas all the other diseases of the bone, however extended they may be, do not lead to such alterations. "It seems

as if it were a peculiarity of otosclerosis which hitherto had neither an analogy nor an explanation." Quite contrary to this, the findings in my experiments show that this new bone formation is *not* a peculiarity only of otosclerosis and that it is *not* confined to this disease.

GENERAL COMMENT

A comparison of the histologic observations on wild birds living in their natural environment with those on wild birds kept in metal cages and on a diet poor in calcium and rich in phosphorus shows the following differences: In adult wild birds living in freedom there is little evidence of reconstruction changes in the bony labyrinthine capsule; only 5 of the 34 animals examined showed a few resorption spaces, which in at least 3 were most likely due to a very mild generalized disease of the skeleton. On the other hand, all the 11 experimental animals showed changes. These changes were always much more marked than those in the wild birds but also were unquestionably manifestations of a generalized disease of the skeleton. The histologic changes were identical with those described by me previously in the chicken capsule. One has every reason to believe that the reactions of the chicken skeleton in general and of the labyrinthine capsule in particular to metabolic or other changes are identical with those occurring in other birds. Under these circumstances I feel that the results of the investigations described, particularly the complete absence of newly formed weblike fibril bone in wild birds living in their natural environment are a sufficient proof that the changes in the chicken capsule do not represent normal processes of internal reconstruction as was assumed by Kamio, Mayer, Meyer and Oesterle. Moreover, the fact that they do not represent normal processes of internal reconstruction and the fact that there was no evidence of venostasis exclude the possibility considered by Meyer that they represent normal processes increased by venostasis. In spite of the fact that the more marked changes in the experimental animals were identical with those found in 6 of the 10 chickens examined by me previously and also identical with those regarded by Wittmaack as manifestations of true otosclerosis caused by venous stasis, it must be strongly reiterated that they do not represent otosclerotic changes. In all the experimental animals the changes in the capsule were associated with a generalized disease of the skeleton, and as long as the contrary has not been proved, there is every reason to assume that also in Wittmaack's animals a generalized skeletal disease was present.

The interpretation of the histologic changes set forth in this paper is entirely different from that of Wittmaack.

According to Wittmaack, the process starts with extreme dilatation of the vessels in the capsule. The surrounding bone is gradually dissolved, the osteocytes are set free, and these proliferate to form a cellular zone around the vessels. The adjoining bone stains reddish because of halisteresis. When the peak of the process is reached, the vessels remain dilated, and the perivascular cellular areas are increased in size, the halisteretic bone is changed in structure, the osteocytes have become larger and much more numerous, the pictures of "sievelike perforation" are frequent, and the red staining has changed into blue owing to reimpregnation with calcium salts.

In the late stage there is a gradual decrease and final disappearance of the venostasis, with increasing fibrosis of the content of the perivascular spaces. The blue staining of the surrounding bone changes into red and, in addition, new red-stained bone is seen on the walls of the resorption spaces.

The residua of the disease are characterized by persistence of relatively large vascular spaces filled with loose connective tissue, which may contain fat cells.

There is a strikingly irregular arrangement of the bony lamellas in the neighborhood of the vascular spaces, apparently owing to the manifold transformations which this bone has undergone.

As already discussed in the introduction, Wittmaack's interpretation is based on a misconception of the basic facts of bone pathology. What he regards as the late stages and residua is histologically identical with the changes described in this paper as those of mild degree. In some cases these changes do not reach a higher degree; in others they are followed by more extensive alterations (of severe degree). The entire blue bone is newly formed weblike fibril bone, and there is no evidence whatsoever of halisteresis. Direct transformation of one kind of bone into another does not occur; sievelike perforation occurs during new formation of very immature weblike fibril bone and not as a sequela of regressive changes in old bone. None of the pictures shown by Wittmaack is comparable with the late stages of human otosclerosis.

In some of the birds described in this paper some areas of weblike fibril bone showed signs of greater maturity as indicated by a larger number and a more regular arrangement of the fibrils, but nowhere had the process progressed to the formation of a breccia which contained lamellar bone. This is all easily understandable if the changes are regarded as due to a metabolic disturbance which at the time of the death of the animals was still active. Under such circumstances one cannot expect to find pictures indicative of a standstill or a healing of the disease. The changes in quails were even more outspoken than in any of Wittmaack's chickens, but there was no evidence of excessive venostasis. One has no reason, therefore, to believe that venostasis is an essential factor in bringing about the bony changes in chickens described by Wittmaack.

The results of the investigation in wild birds confirm the conclusion drawn by me in 1937, based on the examination of chickens. However, in one respect they must be modified. Examination of chickens in which generalized osteodystrophia fibrosa, according to Gohs, had been produced by an injection of a suspension of crushed bone and bone marrow of healthy chickens and of chickens suffering from osteitis fibrosa showed that the changes in the labyrinthine capsule were less marked than in the rest of the skeleton. From this evidence the conclusion had been drawn that though the chicken capsule shows a greater tendency to internal reconstruction than the human capsule, it shows like the human capsule this tendency less than the rest of the skeleton. But this is evidently not the usual reaction. In the experimental animals described in this paper, in which the bone disease had been produced in a more physiologic way, the changes in the labyrinthine capsule were at least equally marked and in some instances even more outspoken than those in the rest of the skeleton. It seems as though the extent of the changes is more or less proportionate to the degree of vascularization of the respective bones.

These present investigations together with those published in my previous paper show that the avian capsule has a greater tendency to internal reconstruction than the mammalian capsule but that the difference in the anatomic structure of the capsule is not sufficient to preclude the use of birds for experiments concerning the genesis of otosclerosis. Thus, the avian capsule should be a particularly good medium for experimentation as it is to be expected that changes can be more easily produced in it than in the mammalian capsule.

The use of the avian capsule for experimental purposes has one important disadvantage. Birds kept in captivity are readily afflicted by a generalized bone disease which may lead to extensive changes in the labyrinthine capsule. In order to avoid misinterpretations of the changes caused by faulty nutrition or other

extraneous factors, extensive and complicated precautions must be taken. Certain bone changes resulting from faulty nutrition are histologically very similar to those seen in the early stages of otosclerosis. These changes apparently were misinterpreted by Wittmaack as true otosclerotic changes.

From a purely theoretic point of view the possibility cannot be denied that an internal reconstruction of the labyrinthine capsule which is histologically similar or even identical with certain stages of human otosclerosis could be produced by venostasis. But the evidence brought forward by Wittmaack in order to prove his claim that the changes described by him were wholly experimental cannot be regarded as sufficient.

It might be added that even if one should succeed in bringing about changes confined to the capsule or to certain parts of it, and even if such changes should lead to ankylosis of the columella, the problem of the causal genesis of human otosclerosis would still not be completely solved. The experiments might elucidate some of the local and initiating factors of the disease, but the constitutional or hereditary factors, which play such an important role, would still remain unexplained. Nevertheless experiments such as I have described should undoubtedly represent an important step forward toward the solution of one of the most puzzling problems in medicine.

SUMMARY AND CONCLUSIONS

In the labyrinthine capsules of various types of adult wild birds, some living in their natural habitat and some in captivity, there was little evidence of internal reconstruction. Quails and pheasants kept in captivity up to nine months on a diet which was poor in calcium and rich in phosphorus showed generalized osteodystrophia fibrosa of the skeleton with certain histologic similarities to von Recklinghausen's type (so-called "low calcium rickets"). The parathyroid glands were markedly enlarged. The changes in the labyrinthine capsules of the wild birds were identical with those frequently found in the chicken capsule. Such changes have been interpreted in various ways by different authors. The changes were identical with those described by Wittmaack in the chicken capsule and regarded by him as experimentally produced.

The changes apparently represent a manifestation in the labyrinthine capsule of a generalized disease of the skeleton most probably caused by faulty nutrition. In spite of their histologic similarity or even identity with certain stages of human otosclerosis, as far as is now known they have nothing to do with true otosclerosis.

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Case Reports

FOREIGN BODY INVOLVING THE FLOOR OF THE ORBIT AND THE ANTRUM

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A case of foreign body involving the floor of the orbit and the antrum is presented because it illustrates the principle that persistence of a draining fistula following apparent removal of a foreign body is highly indicative that a portion

Upper part: patient eleven days after operation, showing the site of the fistula and the incision used; lower part: foreign body removed from floor of the orbit and the antrum.

of the foreign material is still present in the region. Another possibility to be considered is the presence of some form of infectious granuloma in the soft tissues,

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such as actinomycosis. When a persistent fistula is present in the neighborhood of bony structures, formation of a sequestrum or the presence of osteitis or of osteomyelitis may also be suspected. This case also demonstrates the ineffectiveness of the roentgenogram in the diagnosis of foreign bodies which are not radiopaque. The incision used to remove the foreign body illustrates that the approach to the involved region may have to be varied from the routine procedure if the pathologic condition present varies from that usually encountered.

REPORT OF A CASE

A girl 3 years of age was seen on July 13, 1943. In March 1943 she had fallen on a small branch approximately the size of a lead pencil. The foreign body had pierced the skin just below the left inner canthus and had penetrated through the floor of the orbit into the left antrum. A portion of the foreign body had been removed but drainage had continued from a fistulous tract at the point of entrance of the foreign body.

Examination revealed that the fistulous tract led into the anterior ethmoid region and the antrum from above. A moderate amount of mucopurulent discharge was present in the left middle meatus. The roentgenograms revealed clouding of the sinuses on the left with opacity of the left antrum, but there was no evidence of a foreign body. The general physical examination gave essentially negative results.

On July 15, exploration of the floor of the orbit and the antrum was carried out. The incision was made parallel to the lower margin of the orbit (figure, upper part). A probe placed in the fistula led to the roof of the antrum. The periorbita was separated from the bone, and extensive sequestration of the roof of the antrum was found together with a foreign body (weed stem) 5 cm. long and 1 cm. in diameter (figure, lower part), which lay in the floor of the orbit and the roof of the antrum diagonally from above backward and downward, penetrating the ethmoid cells posteriorly. Diseased granulation tissue was removed from the antrum and the ethmoid cells, and after infraction of the inferior turbinate upward a large antral window was made to insure adequate drainage. Crystals of sulfanilamide were placed in the antrum and the ethmoid region. The postoperative course was uneventful. On July 26 the wound was completely healed, and the patient was dismissed. The mild cicatricial ectropion produced will probably require further plastic surgical treatment later.

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Clinical Notes; New Instruments and Technics

USE OF A T TUBE FOR DRAINAGE OF A SEPTAL ABSCESS

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The diagnosis of septal abscess is rarely difficult. The fact that the swelling is not infrequently accompanied by acute sinusitis because of mechanical obstruction of the ostium of a sinus may be overlooked. Incision and drainage of the abscess are commonly done. The problem then arises as to the technic of keeping the wound open and draining well.

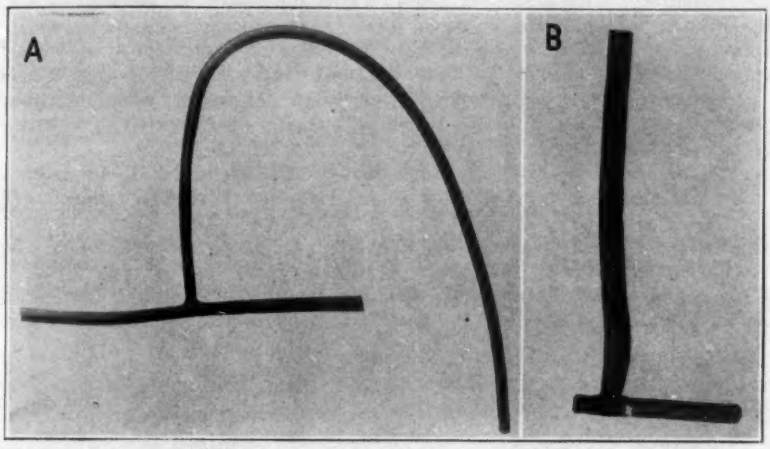

Fig. 1.—*A*, the small T tube which is recommended; *B*, its modification for use.

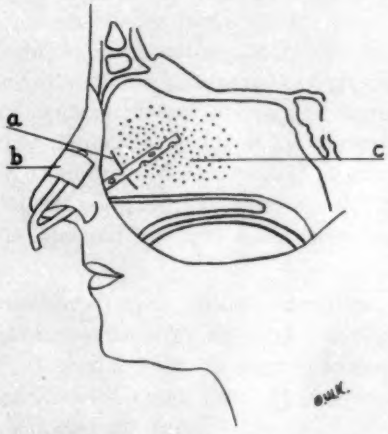

Fig. 2.—A diagram of the drain in situ: (*a*) incision; (*b*) adhesive holding the drain in position on the dorsum of the nose; (*c*) the septal abscess.

A simple method which has been used by us is the employment of a T tube, so well known in surgery of the bile ducts. The fundamental purpose is to have the vertical arm of the tube properly fenestrated and placed in the abscess at the optimum depth, extending beyond the incision to the nasal vestibule. The horizontal arm of the tube then serves a double purpose. Half is fixed to the dorsum of the nose with adhesive tape just lateral to the midline. The upward

direction of the fixed portion automatically provides dependent drainage for the remainder of the tubing. This portion of the horizontal arm is cut short. The secretion from the abscess is easily collected with sterile gauze by the patient, or an external dressing can be devised for this purpose.

A small T tube is selected, cut to the proper length and inserted into the abscess with fenestrations throughout the intranasal tubing. One half of the cross bar of the horizontal arm is fixed to the dorsum of the nose, while the remainder is cut off near the vertical arm. The result is an easily fixed, indwelling, fenestrated, drain with no risk of loss down the throat. Flexibility at the junction of the vertical and horizontal portions of the tubing is a distinct advantage in producing an easy external fixation without traction on the indwelling drain. A simple catheter is less comfortable to the patient and tends to slip out much more easily if it is attached to the dorsum of the nose. The fenestrations combine with the lumen of the tube to secure adequate drainage. The lumen within the tubing can be easily preserved by the introduction of a small silver cannula or a probe into either a fenestration or the external opening at the junction of the vertical and horizontal arms. Similarly, aspiration can be done whenever desirable with a cannula placed within the tubing or beside it where it enters the wound. Because of the resiliency of the drain at its entrance into the wound, there will be found ample room for such aspiration. The tolerance of tissues for this type of drain is well known; T tubes have been left in the bile ducts for many months or much longer without ill effect—a minimum foreign body reaction is produced. Moreover, when the drain is withdrawn after a few days, a fistula results which will remain open much longer than that with many other technics.

Shrinkage of the nasal mucosa is a simple matter and can be done with nasal sprays or drops by the patient himself, for the drain is so small that it does not interfere with such treatment.

Gradual withdrawal of the drain is easily effected by regulating the adhesive tape holding the drain on the dorsum of the nose.

A photograph of the small T tube which is recommended is shown in *A* of figure 1, and its modification for actual use is seen in *B*. Figure 2 is a diagram of the drain in situ.

395 Commonwealth Avenue.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

OTITIS MEDIA AND COMPLICATIONS

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A new publication appeared in 1942, *The Quarterly Review of Otolaryngology*. All available articles in all languages are abstracted promptly and competently. It is a publication for which one should be thankful, although it tempts one to forget that no abstract ever takes the place of the study of the original article.

Another event of great importance to the otolaryngologist was the introduction of penicillin. To quote Crowe and co-workers: [1]

> . . . We otolaryngologists should squarely face the fact that a new era has dawned for our specialty. The sulfonamides, gramicidin and penicillin are only the beginning, but they have worked marvelous cures in many cases which in the past could not have been achieved with surgery alone. Other and even more efficient remedies are sure to follow.

CHEMOTHERAPY

The difference of opinion as to the advisability of using chemotherapy in every case of acute otitis media is still present. Some advise its use in every case, some use it rarely, but the majority reserves its use for the more serious cases.

Richardson [2] in a careful article reports in detail a series of 625 cases of acute otitis media followed from incision to healing. He concludes that the decreased incidence of mastoiditis comes chiefly from the moral support to the surgeon that allows him to defer operation until nature heals the infection. The incidence of mastoidectomy in his series without sulfonamide compounds was only 5 per cent. He states that the 5 per cent incidence of cases of acute otitis media requiring a mastoid operation must be materially reduced by chemotherapy in order to justify routine administration of a sulfonamide compound to every patient with acute otitis media. [A very important contribution proving the point that many patients with involvement of the mastoid process have been operated on too soon and unnecessarily.]

Maxwell [3] strikes a middle ground, using chemotherapy in the following cases: (1) acute fulminating otitis media with signs of mastoiditis on the second or the third day; (2) acute otitis media with virulent infection of the nose and throat; (3) early acute otitis media with much thin discharge under pressure; (4) acute otitis media due to Pneumococcus.

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1. Bordley, J. E.; Crowe, S. J.; Dolowitz, D. A., and Pickrell, K. L.: The Local Use of the Sulfonamides, Gramicidin (Tyrothricin) and Penicillin in Otolaryngology, Ann. Otol., Rhin. & Laryng. **51**:936 (Dec.) 1942.

2. Richardson, J. R.: Observations in Acute Otitis Media, Ann. Otol., Rhin. & Laryng. **51**:804 (Sept.) 1942.

3. Maxwell, J. H.: The Sulfonamide Drugs in the Treatment of Acute Suppurations in the Middle Ear and Mastoid, Surg., Gynec. & Obst. **74**:573 (Feb., no. 2 A) 1942.

Woodward [4] believes that chemotherapy's greatest field lies in the complications of the middle ear infections, in which the mortality has been cut down to a miraculous degree. "Throughout the United States, simple mastoidectomies have dropped to an all time low. This drop started before the advent of chemotherapy and cannot be attributed solely to that drug." [His article contains numerous charts using figures chiefly from the Manhattan Eye, Ear, Nose and Throat Hospital. It deserves careful study.]

DeSanctis, Larkin and Gougelman [5] also give charts of the incidence of otitis media and mastoiditis from 1931 to 1941, covering the study of 1,992 children. There was a great decrease in the incidence of otitis media and mastoiditis in that time. Their conclusion was that sulfathiazole (their choice of the sulfonamide compounds) greatly decreased the incidence of mastoiditis, giving an incidence of 9 per cent, contrasted with 30 per cent in a group not getting chemotherapy.

Bilchick and O'Kane [6] report a study in which 40 patients were treated with sulfanilamide, with 40 controls. The duration of discharge was reduced by one third in the treated group, and the number who underwent mastoidectomy was reduced by over one half.

Morrow [7] reviews the literature on chemotherapy and concludes that chemotherapy should be used early, before destruction of bone, and that the drugs should always be used in large doses.

[It is my custom to give sulfadiazine early and in full doses to all patients with acute otitis media that show by temperature, pain or other symptoms that their infection is more serious than the average.]

Almost all commentators agree that "masking" is a problem which must be met by more vigilance than if chemotherapy were not used. By "masking" is meant the tendency of the sulfonamide compound to conceal symptoms of mastoiditis, such as pain and temperature, and to conceal symptoms of complications such as thrombosis of the lateral sinus.

LOCAL USE OF CHEMOTHERAPY

Currier [8] reports 84 cases in which powdered sulfathiazole crystals were used following simple or radical mastoidectomy. The wound was closed tightly in 24 cases, and in 28 similar cases it was drained with a soft rubber wick. The undrained wounds healed much more quickly. Sulfathiazole was also given before and after operation. The usual daily dose was 1.5 grains per pound (0.18 Gm. per kilogram) of body weight. From 1 to 3 drachms (4 to 11.5 Gm.) was used in the wound. Currier felt that when a drain was used the concentration of the sulfathiazole in the wound was rapidly lost by leakage of blood serum from the wound. [This is a valuable point.]

Guerry and Putney [9] used a gauze drain impregnated with sulfathiazole paste in the mastoid wound in 20 cases; 20 other unselected cases served as controls. In

4. Woodward, J. F.: A Study of Acute Otitis Media and Mastoiditis and Their Relation to Public Health, Virginia M. Monthly **69**:307 (June) 1942.

5. DeSanctis, A. G.; Larkin, V. deP., and Gougelman, W. A.: Otitis Media and Mastoiditis in Children, J. A. M. A. **120**:1087 (Dec. 5) 1942.

6. Bilchick, E. B., and O'Kane, G. H.: Sulfanilamide in Otitis Media in Children, Laryngoscope **52**:341 (May) 1942.

7. Morrow, P.: Chemotherapy in Otitis Media and Mastoiditis, Nebraska M. J. **27**:51 (Feb.) 1942.

8. Currier, W. D.: Sulfathiazole in Mastoidectomy Wounds: Report of Eighty-Four Cases, Ann. Otol., Rhin. & Laryng. **51**:955 (Dec.) 1942.

9. Guerry, DuP., and Putney, F. J.: Local Use of Sulfathiazole in Management of Simple Mastoidectomy Wounds, Arch. Otolaryng. **35**:399 (March) 1942.

the series in which chemotherapy was used the drainage period averaged sixteen and seven-tenths days, 52.8 per cent less than in the control series. Postoperative hospitalization was reduced 55 per cent.

Bryant [10] reports the use of sulfathiazole powder without operation in the treatment of 14 patients, 11 of whom had chronic otitis media with a central perforation and 3 a discharge from the cavity remaining after radical mastoidectomy. Thirteen of the 14 patients with chronic otitis were cured. He also reported a case of acute mastoiditis with phlebitis of the lateral sinus in which powdered sulfanilamide was placed in the wound at postoperative dressings, with cure.

In two good articles Porter [11] states:

> My results from the local use of sprays for acute upper respiratory infections have been disappointing. Sulfanilamide powder has proved more efficient. I am convinced that if they are to be of any value they must, as in the local use of the drug elsewhere, be supplemented by systemic sulfonamides.
>
> We have had limited experience with gramicidin, but so far it has seemed of no help in treating acute staphylococcic infections. For local use in open wounds we prefer sulfanilamide powder used sparingly. The same powdered drug has given excellent results in the treatment of chronic suppurative otitis media.
>
> . . . it was our routine to see that every patient whose case was surgical, with the exception of those undergoing tonsillectomies and minor nasal surgery, was given sulfadiazine to a blood concentration of approximately 10 mgm. per cent.
>
> . . . another factor in the use of the sulfonamides which is of great value . . . is the safety with which surgery can be performed during the acute stages of the disease.

Kolmer [12] in a symposium on chemotherapy advised a dosage of 1 grain per pound (0.12 Gm. per kilogram) per day in severe infections, ½ grain per pound (0.06 Gm. per kilogram) per day in less severe infections, and ¼ grain per pound (0.032 Gm. per kilogram) per day in chronic infections.

> . . . I usually administer at least 5 to 10 grains of sodium bicarbonate with each dose in an effort to prevent nausea and vomiting due to acidosis.
>
> Natural allergy to a sulfonamide compound occurs, but is fortunately rare. It is usually expressed by high fever with skin rashes following the first dose. . . . Fortunately, an individual allergic to one sulfonamide compound may not be allergic to another. . . . A simple and safe test is the oral administration of 5 grains with hourly temperature readings over a period of at least 4 to 6 hours.

He also feels that local use should be combined with oral administration and that the powders are more useful than the solutions. He suggests equal quantities of sulfanilamide, sulfathiazole and sulfadiazine "of such fineness as to pass a 40 to 100 mesh."

Connell and Trowbridge [13] found the powdered sulfonamide compounds of more value than the solutions, using them in mastoid wounds, after nasal operations and after tonsillectomies.

Tobey [14] reviews the literature on the sulfonamide compounds and advises (1) hospitalization if possible, (2) confinement to bed, (3) sulfadiazine to 10 mg. per

10. Bryant, F. L.: Local Use of Sulfanilamide in Otogenic Infection, Arch. Otolaryng. **35**:281 (Feb.) 1942.

11. Porter, C. T.: Practical Uses of Chemotherapy in Otolaryngology, Tr. Am. Acad. Ophth. (1941) **46**:273 (July-Aug.) 1942; Chemotherapy from the Standpoint of the Otolaryngologist, ibid. **47**:34 (Sept.-Oct.) 1942.

12. Kolmer, J. A.: Chemotherapy in Relation to Ophthalmology and Otolaryngology with Special Reference to the Sulfonamide Compounds, Tr. Am. Acad. Ophth. **47**:36 (Sept.-Oct.) 1942.

13. Connell, E. S., and Trowbridge, B. C.: Local Use of Sulfanilamide and Its Derivatives in the Ear, Nose and Throat, Arch. Otolaryng. **35**:740 (May) 1942.

14. Tobey, H. G.: The Sulfonamide Drugs in the Treatment of Acute Otitis Media, Ann. Otol., Rhin. & Laryng. **51**:945 (Dec.) 1942.

hundred cubic centimeters of blood and (4) an arbitrary time limit of seventy-two hours. If progress is not satisfactory, he substitutes sulfanilamide if the culture shows Streptococcus, sulfapyridine or sulfathiazole for Pneumococcus and sulfathiazole for Staphylococcus.

Shaver [15] reports 16 cases in which mastoidectomy was done with use of sulfathiazole powder and complete closure of the wound. Primary union was obtained in 10 cases; all wounds healed within sixteen days.

ACUTE OTITIS AND MASTOIDITIS

Several reviews of large series of cases of acute otitis media and mastoiditis appeared in journals in 1942. Two writers already quoted, Woodward [4] and DeSanctis,[5] commented on the marked decrease in incidence of otitis media and mastoiditis. Woodward believes that much of the credit for this must be given to the better care of children; one factor in this care is the early elimination of tonsils and adenoids; a second important factor is the improvement in knowledge of vitamins and diet.

Priest [16] reviewed the cases of mastoiditis occurring in a ten year period at the University of Minnesota Hospitals. He analyzed in detail the cases occurring from 1936 to 1940. The mortality in the 296 cases was 3 per cent.

Wolf and Capus [17] report 8 cases of "silent" mastoiditis, in which discharge and pain disappear and there is no warning of the infection progressing in the mastoid; Pneumococcus type III is usually present. They warn against the danger of overlooking these cases, a danger especially great when the case is being followed by the general practitioner.

Dixon [18] reports that for five years he has been closing mastoid wounds without drainage and is pleased with the results. He considers "masking" of symptoms by sulfonamide compounds a serious problem.

Neter and associates [19] studied a series of cases of acute otitis media and mastoiditis for the purpose of deciding on the role of Staphylococcus. The use of the plasma coagulase test for determining the virulence of a strain of Staphylococcus is now generally accepted in deciding on the pathogenicity of these organisms. One hundred and six cases of mastoiditis and 438 cases of otitis media were studied over a period of twenty-eight months. Staphylococcus was found in pure culture in 36 per cent of the cases of otitis media and in association with other pathogens in an additional 18 per cent. Of these, about 60 per cent were pathogenic. Staphylococcus was found in only 1 per cent of mastoid sinuses. They point out that pathogenicity does not indicate that the organism is the primary causative agent in cases of otitis media.

Maybaum and Druss [20] report on 73 cases of otitic infection due to Pneumococcus type III. The mortality was 18 per cent. Meningitis developed in 12 cases,

15. Shaver, F. W.: The Use of Sulfathiazole with Primary Mastoid Wound Closure, Canad. M. A. J. **47**:7 (July) 1942.

16. Priest, R. C.: Acute Surgical Mastoiditis, Arch. Otolaryng. **35**:250 (Feb.) 1942.

17. Wolf, G. D., and Capus, B.: Silent Mastoiditis, New York State J. Med. **42**:1755 (Sept. 15) 1942.

18. Dixon, O. J.: Newer Methods in the Management of Mastoid Disease, Texas State J. Med. **38**:394 (Oct.) 1942.

19. Neter, E.; Neuburger, F., and Short, M. L.: Otitis Media and Mastoiditis in Children, Arch. Otolaryng. **35**:631 (April) 1942.

20. Maybaum, J. L., and Druss, J. G.: Otitic Infections Due to the Pneumococcus Type III, J. Mt. Sinai Hosp. **8**:829 (Jan.-Feb.) 1942.

during the first week of infection in 4 of them. Twenty-three cases showed perisinus abscess; 9, epidural abscess; 13, petrositis; 3, labyrinthitis, and 2, abscess of the temporal lobe. They advised that the wound be kept open longer than with the usual organisms and that observation after healing be prolonged.

CHRONIC OTITIS MEDIA AND MASTOIDITIS

Morrison [21] gives a careful description with excellent diagrams of chronic otitis media from the point of view of pathology, with illustrations of the cutaneous flaps to be used in the modified radical operations. Among several points in technic, he advises the use of the Hudson bone drill for removing the cortex of a sclerotic mastoid process. In cases in which intracranial complication is threatened, it is absolutely contraindicated to use the mallet and gouge. The Hudson burr "shortens the entire approach to the mastoid antrum even through solid cortex to two or three minutes and will not harm the dura, the lateral sinus, or the membranous posterior canal wall unless it is grossly abused."

Straus [22] reviews 250 cases of chronic otitis media, in 25 of which radical mastoidectomy was done. Twenty-two of the 25 patients who underwent this operation had satisfactorily dry ears.

THROMBOSIS OF THE LATERAL SINUS

Several interesting articles on thrombosis of the lateral sinus appeared in 1942. Evans [23] reported a case which required ligation of both jugular veins. This is the third reported case in which recovery was observed. The author reviews the literature and states that his patient was studied for a number of years. No bad results followed the double ligation.

McCall and Freeman [24] report a case of thrombosis with recovery following the administration of large doses of sulfanilamide. They review the literature.

Lindsay [25] gives an excellent review of the literature on thrombosis of the lateral sinus and reports 25 cases. The mortality was 24 per cent. He advises chemotherapy with heavy doses followed by adequate surgical treatment.

Singleton [26] reports 6 cases, in 2 of which the jugular vein was ligated. All the patients recovered.

Kemler and Tiefenthal [27] report 3 cases of spontaneous rupture of the lateral sinus. In 2 cases there was spontaneous bleeding from both lateral sinuses, in one seven and in the other thirteen days after operation. One patient required ligation of one internal jugular vein. The literature was reviewed and several cases found in which rupture occurred before operation. These were always fatal.

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21. Morrison, W.: A Modern Conception of the Pathology, Diagnosis, and Treatment of Chronic Suppurative Otitis Media, Ann. Otol., Rhin. & Laryng. **51**:5 (March) 1942.

22. Straus, G. D.: Some Practical Aspects of Chronic Suppurative Otitis Media, Wisconsin M. J. **41**:214 (March) 1942.

23. Evans, M. G.: Bilateral Jugular Ligation Following Bilateral Suppurative Mastoiditis, Ann. Otol., Rhin. & Laryng. **51**:615 (Sept.) 1942.

24. McCall, J. W., and Freeman, M. S.: Septicemia Due to Friedländer's Bacillus, Arch. Otolaryng. **35**:772 (May) 1942.

25. Lindsay, J. R.: Thrombosis of Intracranial Venous Sinuses in Middle Ear Suppuration, Illinois M. J. **81**:467 (June) 1942.

26. Singleton, J. D.: Lateral Sinus Thrombosis: Recent Developments in Treatment, South. M. J. **35**:756 (Aug.) 1942.

27. Kemler, J. I., and Tiefenthal, G.: Spontaneous Rupture of the Lateral Sinus in Otitis Media, Laryngoscope **52**:110 (Feb.) 1942.

Druss [28] reports a case of thrombosis of a cavernous sinus of otitic origin. Autopsy revealed that the cavernous sinus was infected by way of the carotid plexus of veins. Friedländer's bacillus was the causative organism.

MENINGITIS AND ABSCESS OF THE BRAIN

Bradfield [29] reports a case of meningitis following otitis media, inadequate medication having caused "masking." At the time that the diagnosis of meningitis was made, roentgenograms showed that "mastoiditis had cleared." No mastoid operation was done. The patient recovered. [Most otologists would have advised operation.]

Furstenberg [30] states his belief that early intervention in cases of acute mastoiditis and sinusitis is unwise and helps to increase rather than decrease the percentage of cases in which intracranial infection develops. He feels that "masking" is a real danger. Sulfadiazine is his choice of the sulfonamide compounds.

Weinstein [31] reports a study of 39 patients with meningitis of otitic origin who were observed in eight years at the Jewish Hospital of Brooklyn. Of the 10 patients treated with sulfonamide compounds, 3 recovered; the remaining 36 died.

Burman and associates [32] give a detailed analysis of 160 cases of otogenic and 20 of rhinogenic origin; recovery was obtained in 60 per cent of the cases of otogenic and 33 per cent of those of rhinogenic origin. Specific serum and sulfonamide compounds were administered, frequent small transfusions were given and operative treatment was used. This is a carefully prepared article.

Moriconi [33] reported a case of cerebellar abscess in a child of 15, developing after two years of otitis media. The abscess was drained, with recovery resulting.

Hardy [34] reported 3 cases of otitic meningitis with two cures. He stresses the difficulty in locating the source of the meningitis in some cases and the importance of massive doses of the appropriate sulfonamide compound. Sulfapyridine was used in large doses in the cases with recovery.

Asherson [35] suggests that in patients with symptoms of meningitis whose spinal fluid gives no evidence of the disease there is apt to be a lesion in the posterior cranial fossa. If operation does not reveal extradural abscess, the posterior fossa should be explored early. Four cases are reported in detail. His three articles should be studied.

Williams and co-workers [36] review the literature and give the results of treatment for the five years before and since the introduction of chemotherapy. Treat-

28. Druss, J. G.: Thrombophlebitis of the Cavernous Sinus of Otitic Origin, Laryngoscope **52**:115 (Feb.) 1942.

29. Bradfield, S. G.: Meningitis Following Acute Otitis Media Treated with Sulphapyridine, M. J. Australia **1**:529 (May 2) 1942.

30. Furstenberg, A. C.: Intracranial Infections and Their Spread from the Ear and the Nasal Accessory Sinuses, Surg., Gynec. & Obst. **74**:585 (Feb., no. 2 A) 1942.

31. Weinstein, S.: Otitic Bacterial Meningitis, Arch. Otolaryng. **35**:53 (Jan.) 1942.

32. Burman, H.; Rosenbluth, M., and Burman, D.: Otorhinogenic Meningitis, Arch. Otolaryng. **35**:687 (May) 1942.

33. Moriconi, A. F.: Recovery from Cerebellar Abscess of Otogenic Origin, J. M. Soc. New Jersey **39**:385 (July) 1942.

34. Hardy, G.: Otitic Meningitis, Ann. Otol., Rhin. & Laryng. **51**:1062 (Dec.) 1942.

35. Asherson, M.: The Otogenic Abscess, with Special Reference to the Posterior Fossa Cerebrospinal Fluid System, J. Laryng. & Otol. **57**:129 (March) 1942; Cerebellar Abscess: Some Case Records, ibid. **57**:225 (May) 1942; The Otogenic Cerebellar Abscess: Its Operative Treatment and Post-Operative Care, ibid. **57**:281 (June) 1942.

36. Williams, H. L.; Herrell, W. E.; Brown, A. E.; Kernohan, J. W., and Wagener, H. P.: Otitic Meningitis, Laryngoscope **52**:835 (Nov.) 1942.

ment consists of three measures: frequent lumbar puncture, surgical procedure and chemotherapy. Lumbar puncture should be done as often as two or three times daily to keep pressure as near normal as possible. The recovery rate in the five year period before chemotherapy was 35 per cent; this included 20 cases in which cultures of the spinal fluid were not obtained or were negative. The rate is now 69 per cent. In 4 cases meningitis developed one month or more after apparent cure by sulfonamide compounds.

PETROSITIS

Farrior's [37] article presents excellent anatomic illustrations showing a sublabyrinthine approach to the petrous apex. "The most variable factor in this approach is the position of the jugular bulb. In nonpneumatized pyramids the jugular bulb may be superficial and high," blocking this approach. "In a well pneumatized specimen there will be a distance of 5 to 7 mm. between the bulb and the cochlea, room for a very adequate opening into the petrous apex. . . . It is my opinion that such a position of the jugular bulb will exist in any temporal bone in which there is pneumatization of the apex." [This approach depends on the presence of a continuous shelf of pneumatized bone beneath the labyrinth from the mastoid process to the apex. This occurs only in approximately 4 per cent. The operative approach described is limited in practice to this small group.]

ACUTE LABYRINTHITIS

Only one article and one case report appeared in 1942, but both are excellent. Mysel [38] reviews the cases observed at the Massachusetts Eye and Ear Infirmary from 1934 to 1942. A concise review of symptoms is given with a review of the literature. Twenty-two cases were reviewed; there were five deaths. He concludes: There should be no surgical intervention during the period of active labyrinthitis unless an intracranial complication threatens; then labyrinthectomy, not radical mastoidectomy, is indicated. If the acute attack subsides, the surgeon should wait several weeks before doing radical mastoidectomy. The burr must be used; even with the utmost caution, meningitis may be precipitated.

White and Quevedo [39] report a case of acute labyrinthitis precipitated by removal of an aural polyp. This shows again the danger of removing aural polyps. Symptoms of intracranial extension developed, and the labyrinth and cochlea were opened. Recovery was uneventful.

TECHNIC AND TREATMENT

Several excellent articles on the endaural approach for radical mastoidectomy appeared in 1942.

Woodruff and Henner [40] present a record of 28 instances in which the endaural approach was used. They believe that this method offers definite advantages and that they are able to obtain a higher percentage of dry ears than with the postaural approach. Good illustrations are given along with careful data on each case.

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37. Farrior, J. B.: The Sublabyrinthine Exenteration of the Petrous Apex, Ann. Otol., Rhin. & Laryng. **51**:1007 (Dec.) 1942.

38. Mysel, P.: Diagnosis and Treatment of Labyrinthitis Secondary to Tympanic Infection, Ann. Otol., Rhin. & Laryng. **51**:761 (Sept.) 1942.

39. White, L. E., and Quevedo, J.: Chronic Suppurative Otitis Media with Labyrinthitis Following Removal of an Aural Polyp, Arch. Otolaryng. **35**:344 (Feb.) 1942.

40. Woodruff, G. H., and Henner, R.: Endaural Mastoidectomy, Arch. Otolaryng. **35**:777 (May) 1942.

Shambaugh [41] describes and illustrates a new flap for use in the endaural approach. He states that this approach leaves the cavity more accessible to after-care than the postauricular incision.

Leasure [42] advises dropping cleansing or antiseptic fluids into the middle ear and applying pressure with a Siegle otoscope to wash them through the eustachian tube.

Meltzer [43] advocates the endaural approach for the radical and the modified radical mastoidectomy. The postaural approach is reserved for intracranial complications. He thinks that otologic surgeons should give more attention to the preservation of hearing in their operations. Skin grafts in the middle ear tend to cut down hearing. Grafts in the mastoid cavity do not. [I believe that there are advantages in the endaural approach but that no one should attempt it without giving time to a study on the dry bones and on the cadaver.]

Chapman [44] recommends luminous heat in the treatment of acute otitis media. An incandescent light bulb in an ordinary oatmeal or ice cream container answers very well. He also advises the application of this type of heat over the mastoid process following mastoidectomy. [I have had several patients try this, and they have been pleased with the comfort it gives.]

Hershberger [45] states that "many acute ear conditions improve in a few hours after exposure to 5 milliamperes of x-ray at 12 inches for a minute or less."

Titche and Lawson [46] treated 110 patients for acute otitis media by roentgen rays and stated that the average duration of discharge was greatly lessened. No ill effects were seen. The dose should be measured carefully.

Williams [47] writes on the endaural approach, which he advises for radical mastoidectomy. In order to counteract the tendency of the external meatus to contract, he proposes a change in Lempert's incision. The tissue ordinarily excised by Lempert's incision is saved. In the 25 cases in which the technic was used, the time of healing was shortened and a better canal was obtained. The illustrations must be studied.

Kend [48] presents illustrations of flaps to be used in the postauricular modified radical operation. This operation was used in 70 cases with satisfactory results.

Johnson and Solo [49] have recently used the endaural approach in the repair of three postauricular fistulas. The defects were healed within four days. They advise the use of the same approach for radical and for modified radical mastoidectomy.

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41. Shambaugh, G. E.: A New Plastic Flap for Use in Endaural Radical Mastoidectomy, Ann. Otol., Rhin. & Laryng. **51**:117 (March) 1942.

42. Leasure, J. K.: Tympanic Cavity Irrigation, Ann. Otol., Rhin. & Laryng. **51**:253 (May) 1942.

43. Meltzer, P. E.: Hearing in Chronic Otitis Media, Ann. Otol., Rhin. & Laryng. **51**:727 (Sept.) 1942.

44. Chapman, F. J.: Actinic Therapy in Middle Ear Infections, J. Iowa M. Soc. **32**:20 (Jan.) 1942.

45. Hershberger, J. W.: The Treatment of Acute Otitis Media in Children, Pennsylvania M. J. **45**:355 (Jan.) 1942.

46. Titche, L. L., and Lawson, W. E.: Roentgen Therapy in Otitis Media, Am. J. Roentgenol **47**:764 (May) 1942.

47. Williams, H. L.: Antauricular Approach in Radical Mastoidectomy, Proc. Staff Meet., Mayo Clin. **17**:224 (Jan. 14) 1942.

48. Kend, L.: Modified Radical Mastoidectomy with a Tympanomeatal Flap, Dis. Eye, Ear, Nose & Throat **2**:214 (July) 1942.

49. Johnson, L. F., and Solo, A.: The Endaural Repair of Postauricular Fistula, Laryngoscope **51**:469 (June) 1942.

Ide [50] gives a diagnostic test for cholesteatoma; carbon tetrachloride is poured into the ear and allowed to return into a test tube. Enough carbon tetrachloride is added to make 5 cc., and 3 drops of sulfuric acid are added. A green color indicates cholesteatoma. Three cases are reported.

GENERAL CONSIDERATIONS

MacMillan [51] discusses the different roentgenologic positions for proper demonstration of the petrous apex. He gives illustrations of a pneumatic mastoid that underwent complete breakdown and healed by filling in with new bone. The healing occurred without chemotherapy or operation.

Brown [52] reports 2 cases of actinomycosis of the mastoid process. The diagnosis was made only at autopsy in spite of careful examination at mastoidectomy. Any progressive chronic infection of the head or neck should be suspected as a fungous infection and special microscopic and cultural examinations made.

Druss and Allen [53] studied mastoiditis occurring in 49 diabetic persons. In 43 per cent the mastoiditis was caused by Pneumococcus type III. At first this seems to be an extremely high incidence, but it is similar to the 39 per cent incidence in patients of the age of 40 or over reported in another survey at the same hospital, Mt. Sinai, in New York. Destruction of bone occurred earlier than in nondiabetic patients. Another interesting fact was the incidence of postoperative erysipelas in 27 per cent of the patients. This is explained by the increase in sclerosis of the smaller vessels, which resulted in a poor blood supply for the soft tissues and bone. The infections of soft tissues and the necrosis of bone in the mastoid process were more serious in the patients with more advanced diabetes.

Ersner and Saltzman [54] in an evaluation of ear drops found that painful bullas developed in 4 of 10 normal ears treated twenty-four to forty-eight hours with a widely advertised ear drop containing antipyrine and benzopyrine. "At least 35 per cent of our patients develop blebs after use of this mixture." It is therefore contraindicated.

Shea [55] reports 4 cases of fungous infection of the middle ear and mastoid cells.

Trowbridge [56] studied 112 patients with uncomplicated mastoiditis for neurologic signs and symptoms. "Abnormal neurological findings were rare in the series. This is in sharp contrast to the observations of Nielson and Courville in a seemingly comparable series of patients."

TUBERCULOSIS OF THE EAR

Proctor and Lindsay [57] studied 8 cases of tuberculosis of the temporal bone, including the clinical, the general postmortem and the pathologic aspects. They

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50. Ide, D.: Otitic Cholesteatoma with Special Reference to Symptomatology, Eye, Ear, Nose & Throat Monthly **21**:233 (Sept.) 1942.
51. MacMillan, A. S.: Roentgen Findings in Acute Infections of the Mastoid Cells, Ann. Otol., Rhin. & Laryng. **51**:718 (Sept.) 1942.
52. Brown, J. M.: Actinomycosis of the Temporal Bone, Laryngoscope **52**:507 (July) 1942.
53. Druss, J. G., and Allen, B.: Acute Mastoiditis in Diabetes Mellitus, Arch. Otolaryng. **36**:12 (July) 1942.
54. Ersner, M. S., and Saltzman, M.: Ear Drops in Otitis Media, Ann. Otol., Rhin. & Laryng. **51**:471 (June) 1942.
55. Shea, J. J.: Mycosis of the Middle Ear and Mastoid, Laryngoscope **52**:784 (Oct.) 1942.
56. Trowbridge, E. H., Jr.: Neurological Aspects of Simple Mastoiditis, J. Nerv. & Ment. Dis. **95**:575 (May) 1942.
57. Proctor, B., and Lindsay, J. R.: Tuberculosis of the Ear, Arch. Otolaryng. **35**:221 (Feb.) 1942.

present evidence that tuberculosis of the ear is most often a hematogenous spread from a primary complex, similar to that occurring in bones and joints. Photomicrographs of tuberculous lesions in the ear are presented.

Cohen and Koepcke [58] studied 140 adults with tuberculosis of the middle ear. Pain was usually slight or absent, the first symptoms being a sense of fulness and a loss of hearing. Heliotherapy was used in treatment, but benefit was questionable. Twenty-five per cent potassium iodide powder in boric acid applied locally with a powder blower once or twice weekly "helped greatly." Over 50 per cent of the involved middle ears healed without operation. They do not advocate mastoid operation as a routine. They feel that infection usually occurs by extension through the eustachian tube. They use a back rest during pulmonary hemorrhage. Myringotomy is not advised except for relief of severe pain.

Adams [59] noticed that tuberculous otitis frequently followed thoracoplasty, probably because infected material entered an abnormally open eustachian tube. He tested each patient before operation for abnormal patency of this tube by having him blow into a manometer through a glass nasal tip. He was able then to measure the pressure needed before air entered the eustachian tube. If the tube was abnormally open, he inflated the tube preoperatively with a powder containing salicylic acid, in the hope of temporarily closing the tube. [It occurs to me that a postnasal pack inserted before operation might more easily accomplish the same purpose. I have recently seen tuberculous otitis develop following vomiting during a pulmonary hemorrhage, and I think the back rest during hemorrhages should be of value.]

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58. Cohen, S. S., and Koepcke, G. W.: Tuberculosis of the Ear and Mastoid in Adults, Am. Rev. Tuberc. **45**:427 (May) 1942.

59. Adams, J. G.: Tuberculous Otitis Media: Complication of Thoracoplasty, Ann. Otol., Rhin. & Laryng. **51**:209 (March) 1942.

Abstracts from Current Literature

Ear

THE SULFONAMIDE DRUGS IN THE TREATMENT OF ACUTE OTITIS MEDIA. HAROLD G. TOBEY, Ann. Otol., Rhin. & Laryng. **51**:945 (Dec.) 1942.

With regard to the sulfonamide compounds: Tobey discusses six concepts which have been definitely established: 1. They are free to act on infection wherever they can be carried by the blood stream and they will not act where the vessels are blocked by thrombosis, as in the proximity of a localized abscess. 2. The presence or the absence of antibacterial antibodies is important. 3. The toxicity of the drugs is well known and shows most often between the tenth and fourteenth days of administration. 4. About 10 to 15 per cent of patients become sensitized to the sulfonamide compound. If the preliminary medication has failed to stop the spread of the infection, the readministration of the drug, in order to combat any complication which may have arisen later, can cause alarming symptoms. 5. There is a tendency for sulfonamide therapy to mask symptoms in otitis media and mastoiditis. 6. There is a definite but small percentage of deaths connected with the use of sulfanilamide and sulfapyridine.

Otitis media is distinctly seasonal, 75 per cent of the cases occurring, according to Page, in the winter and spring and 25 per cent in the summer and fall. The severity depends on the virulence of the organism which predominates in the prevailing epidemic. The epidemics of infection of the upper respiratory tract vary greatly in their predilection for the middle ear, and there is wide variation in the infecting organisms.

If chemotherapy is going to be successful, there will be a marked change for the better within forty-eight to seventy-two hours, and Tobey suggests that, if it is to be tried, the patient should be hospitalized if possible, and confined to bed. The dosage of sulfadiazine must be adequate to obtain a blood level of at least 10 mg. per hundred cubic centimeters of blood. This will depend on the patient's capacity to sustain a satisfactory blood level. He feels that there should be an arbitrary time limit of seventy-two hours. By this time the cultures made at the time of paracentesis will have been reported on. If the progress is satisfactory, the administration of the drug may be omitted or continued at the same or a reduced dose for a period not exceeding ten days. If the progress is not satisfactory, sulfanilamide for the streptococcus, sulfapyridine or sulfathiazole for the pneumococcus or sulfathiazole for the staphylococcus should be substituted for the sulfadiazine. The continuance of a purulent discharge from the ear after a week of what is considered adequate chemotherapy from the beginning should be evidence that the infection has not been controlled, and continuation of the drug therapy presents grave responsibilities in regard to toxemia, sensitization to the drug and masking of symptoms.

M. V. MILLER, Philadelphia.

SULFATHIAZOLE IN MASTOIDECTOMY WOUNDS. WILBER D. CURRIER, Ann. Otol., Rhin. & Laryng. **51**:955 (Dec.) 1942.

Currier briefly reviews the literature on the use of sulfonamide compounds in mastoid wounds and reports the results in 84 in which sulfathiazole was used. The solubility of sulfathiazole in tissue fluids is ony 150 mg. per hundred cubic centimeters, but this is ten to fifteen times greater concentration than can be obtained by oral administration. As the pathologic process of mastoiditis usually walls off the infection, the absorption is small and the local therapeutic effect is great, and there is no danger of too high a blood concentration from the local use. Currier, as well as others, has been unable to detect sulfathiazole in the blood when the drug was used in mastoidectomy wounds. Currier's investigations have made him decide that sulfathiazole is the drug of choice and that its use in a snugly closed mastoidectomy wound without drain accelerates healing, decreases postoperative drainage and lessens postoperative hospitalization.

M. V. MILLER, Philadelphia.

A HISTORICAL SURVEY OF THE STRUCTURE AND FUNCTION OF THE COCHLEA. T. H. BAST and JAYNE SHOVER, Ann. Otol., Rhin. & Laryng. **52**:281 (June) 1943.

Bast and Shover have made an exhaustive review of the theories of cochlear function. They quote rather fully from the writings of the various investigators and show how the ideas changed and progressed as newer methods of investigation were brought into use. Many

of the observations and interpretations are conflicting, in spite of better laboratory methods, indicating that there is still much work to be done. The paper is interesting and informative.

M. V. MILLER, Philadelphia.

THE PROBLEM OF ACUTE CATARRHAL OTITIS MEDIA. GORDON D. HOOPLE and IRL H. BLAISDELL, Ann. Otol., Rhin. & Laryng. **52**:359 (June) 1943.

The authors review 804 cases of acute catarrhal otitis media occurring over a period of fifteen years. Fluid was found in the middle ear in 419 cases. Treatment consisted of care of infections of the nose and throat when these were found, followed by politzeration or catheterization of the eustachian tube and inflation of the middle ear. Where fluid was found, paracentesis was carried out as well. Some received bouginage of the eustachian tube, some required aspiration of the middle ear by suction, and a small number received roentgen irradiation of the nasopharynx, injections of insulin or thyroid therapy, and various other types of treatment. The number of treatments varied from one or two to frequent ones over a period of months. Of the 804 patients, 15 were definitely not improved.

Of the 804 patients, 666 had infection of the nose or the throat prior to onset of the aural symptoms, and in 8 a contagious disease preceded. Homolateral maxillary sinusitis was present in 44, acute ethmoiditis in 71 and acute sphenoiditis in 1. Acute tonsillitis and infection of the adenoids occurred in 50. One hundred and sixteen showed a septum widely deflected to the affected side.

One hundred patients showed no infection either before or during the course of the aural infection. Allergy was present in 68, hypothyroidism in 19, hypopituitarism in 2, pregnancy in 2, aero-otitis media in 2 and trauma in 2.

They emphasize the importance of determining the predisposing cause. Allergy is the largest factor in the noninfectious type, and if an allergic study does not yield satisfactory results, then endocrine imbalance should be considered. Since most allergics are associated with a low basal metabolic rate, it is not advisable to order a metabolism test on all patients of this group, lest one classify them all as having hypothyroidism. The basal metabolism test plus a sugar tolerance test will differentiate those having a low rate due to allergy and those who really have hypothyroidism.

M. V. MILLER, Philadelphia.

PREVENTION OF EAR DISABILITY. D. A. MCCOY, J. A. M. A. **121**:1330 (April 24) 1943.

McCoy discusses the types of aural damage found in industry and details the decibel losses. Previous remedies are briefly commented on, and a description of the plastic ear mold is outlined, together with the method of how to make such products.

Thirty workers in the California Shipbuilding Corporation who were exposed to noise in the various occupations were fitted with ear molds, and after ten days' use these workers were subjected to questioning, with uniformly gratifying results.

The finished product is relatively indestructible, is a sure block for foreign material, is light and transparent, diminishes intense and high frequency noises and yet permits conversation, and is a die cast reproduction of a plaster model in transparent plastic lucite. It may offer a solution for certain industrial ear problems.

Two figures accompany the article.

GORDON, Philadelphia.

INTERPRETATION OF HEARING TESTS. CHARLES E. KINNEY, Laryngoscope **53**:223 (April) 1943.

Kinney has presented thoughts on the interpretation of the results of hearing tests. Why is it that after inflation of the eustachian tube there is often no hearing gain on the pure tone audiometer and yet definite improvement in hearing the spoken voice? Airplane pilots at 30,000 feet have difficulty in hearing the spoken voice on the radio but hear the pure tone audiometer perfectly. What is the relationship between one's ability to perceive and accurately identify pure tones and one's ability to perceive, identify and analyze the spoken voice?

The author feels that the method of testing with the audiometer should be standardized. He notes that some otologists start with an audible sound and reduce this to where it is last heard, while others start with the tone volume below audibility and increase to the point where it is first perceived. The former method showed better hearing than the latter method, probably because of an effect comparable to the after-image of the eye.

Children with bilateral loss of hearing heard the spoken word better when using both ears than when using one ear, but showed little or no gain when tested bilaterally with the pure tone audiometer.

To get a true picture of one's hearing, the results of the pure tone audiometer and those of the spoken voice test should both be considered. By using a microphone circuit in the audiometer, a more accurate method was devised for testing with the spoken voice. This method as compared with the use of the pure tone audiometer showed that the spoken voice was heard better in cases of conductive deafness, including those of otosclerosis. The spoken voice was heard the same as the pure tone audiometer in cases of uncomplicated otitis media and not as well in cases of perceptive deafness.

The author devised a method (not given) of estimating the percentage loss of hearing, taking into account the loss by both methods of testing. He compares this with the method of estimating loss approved by the American Medical Association. He feels that his method gives a more accurate result.

He suggests that the reason for the difference in perception of the spoken voice and the pure tone audiometer may be that pure tones may be perceived in the cochlea alone, while ability to perceive and analyze the spoken voice lies in the brain itself and has bilateral innervation.

HITSCHLER, Philadelphia.

FACIAL PARALYSIS FOLLOWING MASTOID SURGERY: REPORT OF THREE CASES TREATED SUCCESSFULLY. JULIUS W. MCCALL and FREDERICK S. GARDINER, Laryngoscope **53**:232 (April) 1943.

The facial nerve is usually divided or injured at one of three places: (1) where it crosses the inner wall of the tympanic cavity, (2) at its bend just below the horizontal canal and (3) more deeply in the mastoid process. Injury can be by division or by compression. The latter can be caused by edema, packing or bone chips. The paralysis is the peripheral type. The frontalis muscle is involved too.

After injury, wallerian degeneration takes place: In the distal segment the axis-cylinders degenerate, leaving the sheaths intact, while in the proximal segment the axis-cylinders continue to grow, forming a neuromatous bulb. Ballance and Duel have revolutionized the handling of facial paralysis. An onset of paralysis twelve to twenty-four hours after operation usually means compression of the nerve with physiologic nerve block, while paralysis immediately after operation usually indicates division of the nerve. The faradic current tests the nerve function: A positive response means the nerve is intact. Conservative treatment is then indicated. With no response to the faradic current, immediate operation (nerve graft) is necessary. If the paralysis is of long standing, the facial muscles must be tested (by galvanic current), for if they have atrophied and lost their ability to contract, it is useless to supply them with a new nerve.

The nerve graft is taken from any nerve of about the same size as the facial nerve. The fallopian aqueduct is opened. The ends of the damaged nerve are freshened and then the graft is put in place. It is covered, and the wound is closed. The axis-cylinders of the new nerve degenerate, and the old nerve grows along the pathways provided by the sheaths. Supportive therapy should be given. Function may be regained as early as three months or as late as two years.

Three cases of facial paralysis with successful treatment are presented.

HITSCHLER, Philadelphia.

THE AUDITORY OR EUSTACHIAN TUBE. DOROTHY WOLFF, Laryngoscope **53**:400 (June) 1943.

Wolff presents a microscopic study of 250 pairs of eustachian tubes. She takes up the embryologic development of eustachian tubes.

Measurements of the lumens showed that the eustachian tubes of the infant are not larger than those of the adult. Lack of development of bone surrounding the tubes in the infant allows easier access of the bougie. Because of this, it had been erroneously concluded that the tubes of the infant were larger.

In only a single tube was an organized lymph nodule found. Lymphoid tissue in Rosenmüller's fossa did not effect the closure of a tube in any case.

Otitis media damages the tube. Below the isthmus the tube often shows histologic evidence of a reparative process, but in many cases no such process is observed in the part of the tube above the isthmus.

Only 4 tubes were found to be stenotic. Microscopic sections of the drum membranes corresponding to these tubes did not show excessive retraction. HITSCHLER, Philadelphia.

Larynx

EFFECT OF ANDROGEN THERAPY ON THE VOICE AND VOCAL CORDS OF ADULT WOMEN. JOSEPH L. GOLDMAN and UDAL J. SALMON, Ann. Otol., Rhin. & Laryng. **51**:961 (Dec.) 1942.

In a series of 400 women who received androgen therapy for various endocrinopathic gynecologic disorders there were 23 in whom vocal symptoms of hoarseness, huskiness and masculine pitch associated with structural alterations of the vocal cords resulted from the administration of testosterone propionate. Characteristic was a generalized suffused swelling of the cords with injection of blood vessels running their whole length and a pale off-white color with caudal extension of the swelling so that the subglottic surfaces of the cords could be seen. These changes were constant and, with the vocal changes, persisted in 11 patients for periods varying up to two and a half years after discontinuance of the androgen therapy. It is recommended that the dose be limited to 200 mg. per month to avoid these changes and yet utilize the valuable therapeutic properties of the drug. As an additional safeguard against overdosage, it is recommended that weekly vaginal smears be taken and that the therapy be discontinued if androgenic effects appear in the smear. M. V. MILLER, Philadelphia.

ACUTE LARYNGOTRACHEOBRONCHITIS: TREATMENT OF THE OBSTRUCTIVE LARYNGEAL AND TRACHEOBRONCHIAL EDEMA WITH HYPERTONIC HUMAN PLASMA: A PRELIMINARY REPORT. HARRY L. BAUM, Laryngoscope **53**:371 (June) 1943.

Acute laryngotracheobronchitis often produces death by the inflammatory edema of the subglottic tissues. The violent respiratory effort causes secondary myocardial failure. Tracheotomy reduces the mortality only about 50 per cent and is in itself a somewhat hazardous procedure. After it there may be further changes in the bronchi resulting in asphyxia, atelectasis and pneumonia.

Concentrated plasma has been effective in other edematous conditions including cerebral and nephrotic edema. It is also effective in reducing the subglottic edema of acute laryngotracheobronchitis. This it accomplishes by its high concentration and resultant osmotic action. Such action withdraws fluid from the tissues into the blood stream. It is in no way curative for the infection. It reduces only the edema.

Particularly to prevent overloading of the circulation, preliminary venesection is of value. Blood is removed to a volume three to four times that of the concentrated plasma introduced into the veins. The plasma concentration should be approximately four times its usual strength. This is accomplished by diluting dried plasma with one quarter of the usual amount of water. This procedure is valuable also in the tracheotomized patient, for it prevents subsequent edema. From 24 to 40 cc. of the concentrated plasma is injected, depending on the size of the child. Few reactions are encountered, and these are not severe. If the vein is prepared so that a stylet can be left in it, subsequent injections can be easily administered if necessary. Results are almost immediate. The breathing becomes quiet, the color is restored, and so on. It is to be noted again that the obstruction only is relieved and that the infection has not been cured.

The author suggests other conditions with edema in which this method can be applied. He points out that his own experience has been limited to comparatively few cases. He hopes that physicians interested in the subject will give the method a more extended trial in order that its value may be determined more accurately. HITSCHLER, Philadelphia.

Nose

THE REACTION OF THE NORMAL AND THE ALLERGIC SINUS TO VIRULENT PNEUMOCOCCI. IRA FRANK, MARGERY BLAHD and KATHERINE M. HOWELL, Ann. Otol., Rhin. & Laryng. **52**:81 (March) 1943.

The authors conducted a series of experiments on rabbits, sensitizing them to egg albumin by injecting a 5 per cent solution into the maxillary sinuses. Cutaneous tests were made later to determine the sensitivity. At intervals of three to fifteen days after the last sensitizing injection, various amounts of a highly virulent pneumococcus culture were injected into the sinuses of the sensitized and normal control rabbits. From one to two days after the injection of the pneumococci, the animals were killed, and specimens of mucosa from the sinuses were examined. Nineteen sensitized and 16 normal control animals were used. Two observers made independent estimates of the degree of acute inflammation found, using the number of polymorphonuclear cells as the major criterion. The results found by the two were the same, and the conclusion was that allergy did not alter the severity of the reaction to infection one way or the other. M. V. MILLER, Philadelphia.

A Survey of Antral Surgery: A Review of 100 Cases. Richard H. Stahl, Darrell G. Voorhees and Rector T. Davol, Laryngoscope **53**:186 (March) 1943.

The authors review 100 cases of antral surgical treatment: 50 cases in which the Caldwell-Luc operation and 50 in which antrostomy was performed. They feel that the best way to determine the end results is by postoperative antral washes: Clear washes indicate a cure; purulent washes, a failure. Tabulations have been made of symptoms, related disease, roentgen appearance and other related data. The end results showed 54 per cent of the patients who were treated by the Caldwell-Luc operation and 26 per cent of those who were treated by antrostomy to be cured; in 30 per cent of cases the Caldwell-Luc operation and in 56 per cent of cases antrostomy failed to effect a cure. Reasons are suggested for the high percentage of failures.

Hitschler, Philadelphia.

Cancer of the Nose and Nasal Sinuses. Le Roy A. Schall, Laryngoscope **53**:240 (April) 1943.

Cancer of the nose and sinuses may be primary or metastatic. The symptoms depend to some extent on the site of the lesion. If it is in the nasal cavity, nasal obstruction, particularly unilateral, and hemorrhage are the most usual symptoms. Growths of the maxillary sinus originating in the hard palate or the superior alveolus produce swelling with little or no pain. There may be loosening of the teeth, ulceration and fistulization. If the site is in the posterior superior part of the maxillary sinus, the patient is apt to complain of paresthesia or anesthesia of the cheek and pain referable to the teeth. Such growths can erode into the nose or the orbit or through the anterior face of the sinus. Cancer of posterior ethmoid cells may involve the sphenoid sinus and the optic nerve with resultant diminution of vision. In general, one should always be suspicious of repeated hemorrhage. Biopsy should always be done, and exploratory operation when necessary. A roentgenogram is of aid in diagnosis. It may show a unilateral mass or destruction of bone.

Treatment consists in establishing adequate drainage, irradiating the involved tissue and, often, extensive surgical operation plus irradiation of the involved area. Lateral rhinotomy is used for nasal lesions. It permits exposure of the nasal cavity, ethmoid cells, sphenoid sinus and maxillary sinus. With a tumor of the sinus the approach varies according to the area most involved. If exophthalmos is present, the orbital approach is used after exenteration of the orbital contents; if the lesion is mainly below, resection of the superior alveolus and part of the hard palate is resorted to. For extensive involvement, the author uses a modified Moure incision, by which the whole cheek is turned back. There is no need to ligate the carotid artery.

In any of the aforenamed procedures, the operative cavity should be desiccated with the coagulating current, and radium should be left in to give a dose of from 2,000 to 4,000 milligram hours. The postoperative care is aimed at keeping the cavity clean.

Cancer of the nose or of the sinuses is not hopeless, and the treatment need not be disfiguring.

Hitschler, Philadelphia.

Miscellaneous

Sulfadiazine: Review of Its Use in Treatment of Children. Kenneth J. Winters and Francis R. Janney, Am. J. Dis. Child. **65**:702 (May) 1943.

The hospital records of 115 patients, all unselected, with no "controls," were studied. The ages varied from 3 weeks to 13 years. The conditions ranged from osteomyelitis to pulmonary tuberculosis. The otorhinolaryngologic conditions were acute infections incident to the airways and ears. The authors state that with administration of sulfadiazine high levels in the blood are soon reached and that excretion of the drug is slow; therefore the intervals of administration may be greater and the doses smaller. About ¾ to 1 grain (0.049 to 0.065 Gm.) per pound (0.5 Kg.) of body weight is given over a twenty-four hour period to produce an average concentration in the blood of 7 to 10 mg. per hundred cubic centimeters. Sulfadiazine is effective against hemolytic streptococci, pneumococci, Staphylococcus aureus haemolyticus, meningococci and Haemophilus influenzae. The drug passes readily into the spinal fluid, and the level in the blood can usually be held at two-thirds the aforementioned value. With high levels or with a poor intake of fluids, crystals and red blood cells appear in the urine. Leukopenia and neutropenia clear with discontinuation of the drug.

Patients under sulfadiazine therapy should have regular urinalyses and blood counts. It goes without saying that this program is best carried out when the patient can be hospitalized.

Voorhees, New York.

IMMUNIZATION FOR INDUSTRIAL WORKERS: PROBLEMS OF ORGANIZATION AND ADMINISTRATION. L. D. BRISTOL, J. A. M. A. **121**:816 (March 13) 1943.

The author discusses in moderate detail methods of immunization against a number of diseases and concludes with his views on immunization to infections of the upper respiratory tract:

"If and when the value of protective vaccines or serums against the diseases of the upper respiratory system, such as the common cold and influenza, becomes scientifically established and medically accepted as that of vaccination against smallpox, typhoid and tetanus, it will be early enough for industrial management and industrial physicians actively to promote and encourage such immunizations among workers. This statement is not intended to discourage well controlled experiments or demonstrations, particularly with newly developed products in the possible reduction of sickness absenteeism, lengthy disability and secondary complications and after-effects of colds or influenza."

GORDON, Philadelphia.

METASTATIC BRAIN ABSCESS. ROBERT W. BUXTON and M. L. WHITE JR., Surgery **13**:309, 1943.

In 6 of a series of 132 cases of chronic nontuberculous empyema metastatic abscess of the brain was present, an incidence of 4.5 per cent. In all cases in which the empyema was classified as chronic the known duration was three months or more. In only 1 of the 6 cases was the pulmonary tissue underlying the pleural space free from suppuration. In 4 cases there was no residual empyema cavity at the time of death. Of 72 cases of metastatic abscess of the brain collected from the literature, the cerebral complication arose from an intrinsic pulmonary suppurative process in 82 per cent and was attributed to empyema in 18 per cent. In the authors' cases the cerebral condition ran an acute course, in 4 cases the period from the onset of cerebral symptoms to death being five to ten days. None of the abscesses were encapsulated; in 4 cases the lesion was solitary, in 1 case multiple and in 1 case multilocular. In 2 cases the abscess ruptured into the ventricles, and in all cases purulent meningitis was present. No definite correlation was noted between manipulation at the site of drainage of the empyema cavity and the onset of cerebral symptoms. In 2 cases the cerebral abscess was drained surgically, and in 1 the abscess was evacuated through the exploring needle. In these 3 cases appropriate use of sulfonamide compounds was carried out, but all the patients died.

SHENKIN, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

THROMBOPHLEBITIS OF THE CAVERNOUS SINUS: RECOVERY. A. R. C. DOORLY, Brit. M. J. **1**:42 (Jan. 9) 1943.

A healthy married woman aged 56 had a nasal furuncle inside the left nostril. There was the usual edematous swelling of the adjacent cheek, with intense pain and slight fever. She was treated with sulfathiazole, one tablet (0.5 Gm.) every four hours, and glycerin packs were advised for the interior of the nose. On the following day there was more swelling, the temperature was 101 F. and pus was being discharged from the furuncle. Two days later the left eye was proptosed, both lids were greatly swollen and the right eye was commencing to be involved. The patient complained of seeing double on looking to the left. There was some rigidity of the muscles of the neck, and the temperature rose to 103 F. A blood culture showed Staphylococcus aureus of the same type as that found in the nasal lesion. She was treated with transfusion of 400 cc. of whole blood and intramuscular injection of sulfathiazole alternating with oral administration of the drug, 4 Gm. being given by each route within the first twenty-four hours. After two days the temperature began to fall and the general condition improved. The chemotherapy was continued, though after three days the dose was reduced. The next day there was a sharp rise of temperature, and a second transfusion of blood was given. This was followed by improvement, but there was a third relapse. Another blood transfusion was given and the sulfathiazole treatment continued. During the next four days the patient remained very ill, with an extensive rash and severe headache, but the local condition of the eyes showed improvement. As the rash was so distressing and the patient by then had taken 95 Gm. of sulfathiazole, the chemotherapy was discontinued. After this the temperature gradually came down, the rash faded and the swelling left the lids. Headache gradually ceased, and convalescence began.

It has been generally recognized that free incision is dangerous and apt to lead to rapid dissemination of the infection. Hence the method of treatment reported, offering a good chance of recovery, is extremely gratifying. It is difficult to say whether the blood transfusions or the sulfathiazole played the greater part in the cure of the condition, but the author

thinks the combination important. The case proved conclusively the great value of sulfathiazole in the treatment of serious staphylococcic infections and showed that the toxicity of this drug is only half that of sulfapyridine.

KNAPP, New York. [ARCH. OPHTH.]

MEMBRANOUS INFLAMMATION OF OROPHARYNX, NOSE AND CONJUNCTIVA DUE TO SULPHATHIAZOLE ADMINISTRATION. I. M. SCHNEE, Brit. M. J. 1:506 (April 24) 1943.

The following case is reported as evidence of an unusual type of toxic reaction to a sulfonamide compound. The reaction consisted in inflammation involving the conjunctiva, nose, pharynx, mouth and larynx, with the development of a membrane. There was rapid recovery on discontinuance of the chemotherapy.

The patient, a soldier 28 years of age, had been treated with sulfathiazole for an infection of the upper respiratory tract. After he had received about 18 Gm., the symptoms seemed to be relieved, and treatment was stopped. On the fifth day of chemotherapy the eyes became inflamed and a discharge was present; then the mouth became sore and the lips cracked. The fever recurred and the patient was sent to the hospital. The lids were swollen, with a smooth, thick, well formed gray membrane which left no bleeding surface when it peeled off but reformed within a few hours. The cornea was not involved. A similar condition was found in the mouth and throat. Smears and cultures were negative except for staphylococci. On the patient's relapse, sulfathiazole treatment was renewed. When the condition of the eyes and throat became worse, at the suggestion of Lieutenant Colonel Derrick T. Vail use of the drug was discontinued. The patient rapidly became well.

KNAPP, New York. [ARCH. OPHTH.]

DEFECTS OF SMELL AFTER HEAD INJURY. A. D. LEIGH, Lancet 1:38 (Jan. 9) 1943.

Traumatic impairment of the sense of smell received relatively little attention until recent years. Ogle described 3 cases of anosmia consequent to injury of the head. He stated that there could be little doubt that this loss was due to rupture of the olfactory nerves as they pass from the olfactory bulb through the ethmoid bone. Goland (1937) refuted this statement and suggested, in agreement with Legg (1873), that the loss results from damage to the lateral olfactory stria and tip of the temporal lobe by impact against the lesser wing of the sphenoid.

Of 1,000 patients representing consecutive admissions for head injuries to a military hospital, 72 (7.2 per cent) had impairment of the sense of smell. Simple clinical tests were used. The test substances were coffee, camphor, eucalyptol, peppermint and clove. An airway free of blood and mucus was considered satisfactory. A single sniff up each nostril replaced the technic of Elsberg and Levy (1935). Of the 72 patients, 2 had gunshot wounds, 2 died, 41 had complete anosmia, 31 presented general diminution of olfactory acuity or unilateral anosmia and 12 complained of parosmia. In 18 of the patients the injury was occipital (in 12 associated with fracture); in 30, frontal (in 27 associated with fracture), and in 7, parietotemporal, and the site of injury in the remaining 17 patients was not determined, although 10 of these had fractures. Five of the occipital fractures had fissures into the foramen magnum, and 14 of the frontal fractures involved the frontal sinus, with rhinorrhea in 5 cases.

The violence of the injury was usually extreme, as shown by the high incidence of organic neurologic signs. The post-traumatic amnesia was reckoned in days for all but 6 patients. Twenty-six men were so incapacitated that they were discharged from the Army (36.4 per cent of this group, as compared with 29.6 per cent of the entire group of 1,000 patients). Only 34 men returned to full duty, and 12 were returned as fit for light work only.

In only 6 of the 72 patients has any recovery of smell been noted, the data on these men being tabulated as follows:

Site of Violence	Parosmia	Time of Recovery
Crush	Yes	20 days
Frontal region	Yes	3 mo.
Occipital region	Yes	12 mo
Crush	No	4 mo.
Frontal region	No	3 mo.
Occipital region	No	1 mo.

Parosmia was observed in 12 patients. Two of these men experienced unpleasant smells spontaneously, and in the remainder the parosmia was associated with olfactory stimulation. The type of smell was nearly always unpleasant. The onset of post-traumatic parosmia was

usually delayed from seven days to three months. Of the 6 patients who recovered, 3 experienced parosmia for a period, and the question arises as to whether this is part of the recovery cycle.

In 2 patients loss of taste and loss of smell, with subsequent recovery, went hand in hand. Most patients had no disturbance of taste. Of 41 with complete anosmia, only 6 complained of change in taste. Of the entire group of 72, only 14 complained of disturbance in taste. In 9 of these 14 patients primary tastes were preserved and only appreciation of flavors was lacking. In 2 patients taste was perverted. Six of the 14 patients had parosmia. The results for this group of patients indicate that the connection between taste and smell is not so close as has been popularly supposed.

The inconstant association of the loss of taste and the loss of smell in most cases is not fully explained by the clinical observations. Perhaps taste is more affected by olfactory stimuli in some persons. In addition to the tearing of the olfactory filaments, there may be damage to the temporosphenoid lobe or to the region of the anterior perforated crater. Also, taste fibers may be concomitantly affected in their course through the petrous bones. Seven of the 14 patients with disturbance in taste showed a direct fissured fracture from the parieto-temporo-occipital region toward the base and had other signs of possible petrous damage.

SANDERS, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

PNEUMOCOCCIC MENINGITIS IN CHILDREN AND ITS TREATMENT. GRETA MUHL, Svenska läk.-tidning. **38**:1284 (June 6) 1941.

Muhl presents a report of 28 cases of pneumococcic meningitis in infants observed between 1926 and 1938. Most of the patients were treated with ethylhydrocupreine hydrochloride. Seven of the infants recovered. In addition 3 new cases of this condition in infants are reported, in each of which recovery followed treatment with sulfapyridine.

WALLGREN, Göteborg, Sweden. [AM. J. DIS. CHILD.]

News and Comment

CASSELBERRY AWARD OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION

A sum of money having accrued from the Casselberry Fund of the American Laryngological Association, a prize will be offered in 1944 for papers on original investigation in the art and science of laryngology or rhinology. These must be in the hands of the secretary, Dr. Arthur W. Proetz, 1010 Beaumont Building, St. Louis 8, Mo., before March 1, 1944.

Society Transactions

LEGE OF PHYSICIANS OF PHILA-ELPHIA, SECTION OF OTOLARYN-GOLOGY, AND PHILADELPHIA LARYNGOLOGICAL SOCIETY

GABRIEL TUCKER, M.D., *Chairman*

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THOMAS F. FURLONG JR., M.D., *Secretary*

Regular Meeting, April 21, 1943

EORGE L. WHELAN, M.D., *Editor of Abstracts*

orrelation Between Vestibular Function and earing in the Profoundly Deaf Child. Pre-nted by DR. MARY FRANCES VASTINE (by invi-tion).

is article will be published in full in the ARCHIVES TOLARYNGOLOGY.

DISCUSSION

. WALTER HUGHSON: Dr. Vastine has presented esults of her research in a scholarly and concise er. She has given precise and accurate data on a em which has not before been studied with even pproximate degree of thoroughness. She has estab-d a specific relationship between vestibular and tic function in the group of deafened children consideration. Her findings are immediately cable to all similar groups, of which there are rtunately many in this country.

the outset I should like to sound a note of warn-elative to her quotation from the late Dr. Gold-s book, "The Problem of the Deaf." His state-that "though the acoustic labyrinth does not nd to functional test, the histological elements that rise it may not be entirely beyond salvage if the gmus reaction still be present," holds out, I feel, njustified hope. Since the advent of modern metry, no evidence has been presented to indicate a damaged organ of Corti can be salvaged by any od now available.

wever, when residual hearing is present, and this the case in 3.2 per cent of the ears Dr. Vastine ined, it can be utilized by modern pedagogic ics.

fortunately, we have few opportunities to study logic material in cases of embryonic deafness. In ohns Hopkins University collection of serially sec-d temporal bones there is no single specimen relat-o this condition. That fact probably eliminates ossibility of studying sections of such ears which previously been examined by accurate audiometric ods.

e aural changes in cerebrospinal meningitis are a r of recent record, and the wonder is that Dr. ne found any remaining labyrinthine activity in 7 per cent of ears so affected. It is difficult to stand how so fulminating an infection could take and any part of the neural mechanism of the ear survive.

m not sure that I can agree with Dr. Vastine's nation of the "cause unknown" group as being a whose deafness is due to some postnatal etiologic factor. Many parents state that the child had hearing in infancy but never acquired speech. My co-workers and I have arbitrarily set speech, if only in single words, as a criterion for hearing in infancy. Children without such a specific history must be considered to have been born deaf. The other listed causes of deafness are fairly evenly distributed through the general group of contagions and infections. Syphilis is a relatively rare etiologic factor, as Ciocco pointed out several years ago. This is based not only on histories but on the number of children with positive Wassermann reactions in the total school population. At this point may I draw attention to mumps as an etiologic factor in the complete destruction of the static and acoustic labyrinth? The damage is frequently unilateral and hence may go unrecognized for months and even years, and of course a child with this type of deafness never qualifies for admission to a school for the deaf.

I mention that Dr. Vastine's interpretation of her method of carrying out the caloric test for vestibular function might be questioned only to say that her results permit of no questioning. When the correlation of vestibular and cochlear function falls into a pattern so succinct one must feel rather that another means is now available for the measurement of auditory integrity.

No purpose would be served in reviewing in this discussion the careful and convincing statistical analysis which has been presented. The tables and charts represent a long and painstaking research effort.

The summary of the findings is highly significant in several respects, chief of which is the fact that 81 per cent of the ears examined exhibited some degree of vestibular activity as determined by a completely objective test. This lends strong support to the subjective finding of residual acoustic activity in an even higher percentage of profoundly deafened children. The fact that a "type audiogram" can be drawn up corresponding to th vestibular activity of the ear is an entirely new and significant finding.

Embryonic deafness has been one of the completely blank spots in the pathology of hearing. Dr. Vastine's work is an important contribution toward the ultimate clarification of this problem.

DR. GEORGE M. COATES: I want to compliment Dr. Vastine on this fine piece of work that she has done. She has been fortunate in having access to such material as that in the Pennsylvania School for the Deaf.

Dr. Hughson mentioned that this work is extremely important and throws light on subjects otologists do not know too much about. Dr. Hughson has covered this whole paper so well in his discussion that not much is left for me to say. Again, I want to thank Dr. Vastine for her excellent contribution.

DR. DOUGLAS MACFARLAN: This is a most comprehensive and useful study, bringing into exact form the evidence that certain proportions of vestibular damage can be expected in the deaf child. Dr. Vastine's paper infers that otologists can surmise with some accuracy the status of the damage to hearing and the possibility of the presence of usable hearing. The latter item is most important from a pedagogic point of view.

The group that was tested was made up of young persons 14 to 16 years of age. I wish to plead for vestibular testing in the small deaf child who is brought to the otologist when deafness is first suspected—aged 2, when speech shows no signs of beginning. One must decide whether there is sufficient residual hearing to

warrant the use of amplified sound to teach these children language. Too often "deaf traits and habits" develop early which later have to be overcome. This is particularly true of auditory inattention in those considerably deaf but with usable hearing.

I have been testing the hearing of such children with the conditioned response technic, and invariably I put them through a fractional caloric test under the lightest anesthesia. You will remember that the quick component of the nystagmus disappears under anesthesia, but as the child is coming out the true deviation (the slow component) appears. The eyes roll over and stay over. After one ear is tested, a period of rest and recovery is allowed before the opposite side is tested. Testing a child under anesthesia is the only possible way of conveniently and accurately studying restless children. Further, it gives an opportunity for a good otoscopic examination and for an examination of the nasopharynx and the patency of the eustachian tubes.

DR. BENJAMIN H. SHUSTER: There is no doubt about the importance of Dr. Vastine's work. I also want to add that much credit is due her for publishing these records. About fifteen years ago I did some similar work on 100 children in a clinic for deaf persons. I believe there is a summary of these cases in the archives of the Board of Education. The findings were more or less similar to Dr. Vastine's. I cannot recall the figures accurately. The children whose deafness was congenital often had good vestibular responses. I examine infants in my office right along. The caloric test has not been accurate, as children do not cooperate. I usually seat the mother in a rotation chair and she holds the child's head tight against her chest and I observe the responses after rotation. I wish to congratulate Dr. Vastine on this excellent summary. I hope it is published where it will be appreciated.

DR. MARY FRANCES VASTINE: I want to thank everybody for the generous discussion.

A Review of the Etiology of Ménière's Symptom Complex. Presented by MR. E. DOWNES LONGAKER (by invitation).

In this paper an attempt is made to mention briefly some of the more important theories which have been brought forth concerning the causation of Ménière's symptom complex.

In 1861 Ménière described a recurring symptom complex consisting of severe attacks of vertigo, nausea and vomiting, and tinnitus, these being accompanied by progressive loss of hearing. He stressed the recurrent nature of the attacks and the well-being of the patient between them. Moreover, Ménière recognized the condition as one of the ear rather than the brain, pointing out that it differed from a cerebral accident, there being no paralysis, that the patient was well aware of what had occurred and that the only mental sequela was the fear of a subsequent attack.

In addition, Ménière presented the results of an autopsy on a young woman who died within twenty-four hours after the onset of an attack. Finding nothing in the brain to explain the clinical condition, he sectioned the temporal bone and discovered a sanguinous material in the semicircular canals. Since that time there has been much speculation as to what might have been the cause of the hemorrhage. In the light of present knowledge this case was apparently one of the rarer ones of what physicians today designate as Ménière's symptom complex.

Through the years, much evidence has been brought forth which implies that a great many diseases can cause or be accompanied by this symptom complex. However, there is reason to believe that, if Ménière's symptom complex is defined as a recurring phenomenon consisting of progressive deafness, tinnitus, vertigo, nausea, vomiting and spontaneous nystagmus, many of these may have been erroneously included.

No less a man than Charcot was the first to suggest, in 1874, that division of the nerve might be used in the treatment on Ménière's disease, since most patients were apparently cured when the deafness became complete. In 1928 Dandy, reporting results of this procedure, brought forth still other theories than those mentioned to date. He pointed out that "in the symptom complex expression of Ménière's disease one is strongly reminded of other human ills, such as trigeminal and glossopharyngeal neuralgia and possibly epilepsy." He further stressed the facts that each shows periodicity, that pain comes without warning or apparent cause and that each suggests lesions of nerves or nerve tracts. Several years later he said, somewhat dogmatically, "We believe attacks of Ménière's disease are exactly similar to the paroxysmal attacks of trigeminal and glossopharyngeal neuralgia."

These speculations are undoubtedly interesting, but to say that Ménière's disease is caused by the condition which causes trigeminal neuralgia is to say in a roundabout way that one does not know the cause.

The evidence for focal infection as a cause of Ménière's disease consists of reports of cure after removal of such foci—the cure of a disease which is known to have asymptomatic periods and from which there is not infrequently spontaneous recovery as well. However, it must be admitted that much of this information comes from reliable investigators, many of whom are outspoken in their belief that the removal of such foci is of much benefit. In all cases previously reported by Hallpike and Cairns and Hallpike and Wright in which autopsies were done some form of focal infection was demonstrated.

And, finally, Shambaugh and Roberts in their report stated that "in the majority of cases the disease is one with serous labyrinthitis from focal infection or an allergic labyrinthitis."

As with focal infection, there is much evidence in the form of "cures" that points to allergy as one of the causes of Ménière's disease. As just mentioned, Shambaugh and Roberts feel it plays an important part in some cases. Moreover, many cases are described in the literature in which a person found to be allergic to a certain substance gets complete relief on withdrawal of the offending allergen and is perfectly well until the substance is again encountered.

And so having briefly reviewed some of the various etiologic theories of what is best designated as Ménière's symptom complex, the question arises—Is there an underlying mechanism common to them all? One may not say, but the relation of water balance to edema, the relation of allergy to bacteria, the relation of histamine to allergy and the relation of histamine to local edema all seem to point to the fact that many things may act through a common underlying mechanism, the cause of the symptom complex first described by Ménière.

DISCUSSION

DR. WALTER HUGHSON: Mr. Longaker's review of the literature on Ménière's symptom complex is extremely interesting and comprehensive. It is quite true that Dr. Dandy's approach is a purely surgical one, and this can be readily understood when the size of the series of operative cases is considered.

Recently Guild, of Baltimore, has had an opportunity to study the temporal bones in a case of Ménière's disease, and his sections amply confirm the findings of Hallpike and Cairns reported some time ago.

g feature of the pathologic picture being marked
ion of the entire endolymphatic sac and practical
ation of the scala vestibuli.

phase of the problem which Mr. Longaker has
uched on is the striking effect of seasonal and
pheric changes on this disease. By careful study
patients it is often possible to predict a recurrence
mptoms on the basis of changes in barometric
re. In considering the effect of any form of
y, these meteorologic factors must always be
into consideration.

THOMAS F. FURLONG JR.: The term "Ménière's
" is now commonly used to designate the clinical
of recurrent attacks of vertigo associated with
y disturbances. The association of this condition
definite pathologic entity has provided a more
basis for therapy.

bition of Hearing Aid Collection in the Mutter Museum. Presented by DR. DOUGLASS ACFARLAN.

re are many things of interest to the otolaryn-
st in the Mutter Museum. There is, for example,
rge collection of sinus specimens made by Dr.
ew Cryer, one of the first and finest collections.
o be seen is a beautiful plaque containing, each in
all square, the auditory apparatus of the verte-
. One should read Max Goldstein's articles on
arative anatomy of the ear, then study these speci-
There is a collection of illustrations of Fraser's
tions of the mastoid process and middle ear
he laryngologic instruments of the pioneer Ameri-
aryngologist, Dr. Solis Cohn, and this collection
exhibited shows the evolution of the hearing aids.
eady there are a number of such exhibits else-
. The Army Medical Museum houses the collec-
made at the time of the first survey of hearing
in 1924. The work at that time was done under
spices of the National Organization of the hard-
ring. The purpose of the study was to eliminate
orthless aids being sold and to advise deafened
ns on all aspects of the subject of aids. This work
w in the hands of the capable committee of the
ican Medical Association, whose efforts have
d fruit in standards set up for both audiometers
ids. The tactful and efficient secretary, Howard
rter, is supported by a selected group of otologists
ularly familiar with the problems. Philadelphia
resented by three members—Drs. Coates, Hewson
Macfarlan, all members of the societies meeting
t. A sample of a committee release is shown.
Max Goldstein accumulated an excellent collec-
of hearing aids, which is now in the Central
ute in St. Louis; another good collection is that
New York League for the Hard-of-Hearing.
re is shown a group of nonelectric aids, the horns,
s, tubes and other types of sound collectors, ex-
t in their day, free from distortion effects, but not amplifying greatly. Besides this, they are unusually conspicuous and cumbersome. Next is a collection of prostheses—artificial drumheads of various types, the original being Loyabe's drum, a circular piece of parchment with a silk strand threaded to the center. Some of these drumheads are still being used to overcome the effects of a permanent perforation, they are not to be considered free from danger when left in too long or when discharge is present.

Next exhibited are the so-called carbon aids, which are adaptations of the original telephone principle. Many different models of these have come and gone on the market. Some used multiple microphones. Greater amplitude was gained, but with greater distortion. A graph is presented showing the "response-characteristics across-the-frequency-range" of a number of different carbon aids; one may notice the peaking in the region of 1,000 cycles. Also a midget receiver is shown, and an early bone conduction receiver.

The next step in the evolution of aids came with the advent of the "peanut" audion bulb, which was first developed by the Thomson-Houston Company in England. This made possible the portable "tube sets" now supplanting practically all other types. "A" and "B" batteries have to be used, but all have been reduced in size and weight. There previously had been a non-portable tube aid on the market, before the "peanut tube" made the portable type practical. This aid was produced by a deaf man, E. A. Myers, whose microphone made the best showing in the first survey.

With the appearance of crystal receivers and microphones, another step forward was made. The principle of the use of the crystal was a revival made possible by progress in amplification. Quality and fidelity are much better with the crystal, which is usually stable and dependable. It is interesting to note that Dr. Fowler, of New York, worked on this idea many years ago with the thought of developing something worth while in bone conduction.

A recent acquisition is the original oscillator used by John Gutman for testing hearing. This instrument is the father of the present frequency audiometer, and herewith is presented the first model of the conditioned response trainer.

I cannot leave the subject of hearing aids without voicing a plea for a better understanding of aids by otologists. The public expects it of them, and the deafened people come to them for particular information on aids. Otologists must at least be able to advise these people. They can help them make an intelligent selection by comparative interpretation tests with the different aids which are being considered by them. At the same time otologists must warn them to safeguard their hearing. Otologists must learn to appreciate the trials and tribulations of the retail dealer in handling hearing aids; he has not an easy path with deaf persons, many of whom expect too much from an aid. Otologists and dealers may learn much from each other and can help each other greatly; all to the ultimate benefit of the handicapped.

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